NERVE CONDUCTION STUDY & EVOKED POTENTIALS

IN VISUALLY CHALLENGED PERSONS

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CERTIFICATE

This is to certify that the dissertation entitled **"NERVE CONDUCTION STUDY & EVOKED POTENTIALS IN VISUALLY CHALLENGED PERSONS"** is a bonafide original work of **Dr.V. Sadeesh kumar,** in partial fulfillment of the requirements for D.M. Branch– I (NEUROLOGY) Examination of the Tamil Nadu Dr. M.G.R Medical University to be held in AUGUST 2013, under our guidance and supervision.

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DECLARATION

I hereby solemnly declare that this dissertation titled **"NERVE CONDUCTION STUDY & EVOKED POTENTIALS IN VISUALLY CHALLENGED PERSONS"** was done by me in Institute of Neurology, Madras Medical college and Rajiv Gandhi Government General Hospital, Chennai -3, under the guidance and supervision of **Prof.K.BHANU,Dip.N.B(MED).,D.M.,** Professor of Neurology, Institute of Neurology, Madras Medical College & Rajiv Gandhi Government General Hospital, Chennai. This dissertation is submitted to the Tamil Nadu Dr. M.G.R. Medical University towards the partial fulfillment of requirement for the award of D.M Degree Branch I (NEUROLOGY).

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CONTENTS

INTRODUCTION

The development and refinement of instrumentation has been a great asset in the diagnosis of neurologic diseases. First available study was in 1951 by GALLEGO A.[1] Peripheral nerve conduction study is an extension of neurological examination. Data are available for normal persons and how it gets changed in diseases process. From 1975 onward numerous studies are published regarding normal data, techniques, methods and origin of waves etc.

Peripheral nerve conduction studies are done in very few of the visually challenged. PubMed advanced search of "Somatosensory Evoked Potentials" and "visually challenged persons" yields no available data.

Vision

Eighty percent of the surrounding information is appreciated through visual sense. So vision takes the priority among the all senses. Quality of life gets affected when the visual sensory modality is less or absent. Persons with low vision having much difficulty in day to day activity. The risk of physical and social isolation is greater for people who are blind. Some causes of blindness can't be prevented.

When a person has a visual impairment, getting information about the environment and moving safely in the environment can present the need for alternative techniques and creative strategies. They use touch and hearing for locomotion in environment.

Blindness:

Refers to a condition where a person suffers from any of the following

conditions namely,

1) Total absence of sight, or
2) Visual activity not exceeding 6/60 or 20/200 (snellen) in the better eye with correcting lenses, or
3) Limitation of the field of vision subtending an angle of 20 degree or worse.

This definition is incomplete as it inadvertently omits quantification of the acuity as well as the field of vision as is done in the case of the WHO definition.

Categories of visual impairment				
Category	corrected VA (better Eye)	WHO definition		Indian Definition
		Standard	Working	
0	6/6-6/18	Normal	Normal	Normal
1	<6/18-6/60	Visual impairment	Low vision	Low vision
2	<6/60-3/60	Severe visual impairment	Low vision	Blind
3	<3/60-1/60	Blind	Low vision	Blind
4	<1/60-PL	Blind	Low vision	Blind
5	NPL	Blind	Total Blindness	Total Blindness

Many studies are done in visually challenged persons analysing the event related potentials and for cortical neuronal plasticity. Studies analysing the conduction time in somatosensory evoked potentials are lacking. Number publications on evoked potentials are decreased after the widespread clinical utility of Magnetic Resonance Imaging. We studied the visually challenged persons nerve conduction and evoked potentials.

AIM OF THE STUDY

1. To study the peripheral nerve conduction in visually challenged persons.
2. To study auditory and somatosensory evoked potentials in visually challenged persons.
3. Comparison of previous available data with our control population.

REVIEW OF LITERATURE

Visual Acuity

Visual acuity is the measurement of the ability to discriminate two stimuli separated in space at high contrast compared with the background.[2]

The visual acuity for distance is measured as the maximum distance at which person can see a certain object, divided by the maximum distance at which a person with normal eyesight can see the same object. Thus a visual acuity of 6/60 means that the person examined cannot see, at a distance of 6 meters, the object which a person with normal eyesight would be able to see at 60 meters.

The simplest method of testing visual acuity is to see whether the person can count fingers at a distance of six meters.

Neuronal plasticity

The investigation of neuro plasticity has expanded rapidly over the past ten years and has uncovered a remarkable capacity of both the juvenile and the adult brain to be shaped by environmental input.

In particular, a wealth of studies has documented striking effects of sensory deprivation in one modality on the development of the remaining modalities.

Individuals who become blind during the course of their lifetime have to adjust to the striking demands of their environment. The brain areas which are used for vision is recruited in a compensatory cross model manner to other modalities of sensation like touch and hearing.

It is a common belief that the loss of vision is somehow compensated for by the remaining senses.[3] Increased performance in the other sensory modalities observed when visual sensation is deprived off through brain reorganization as compensatory changes.

For their learning, reading Braille is used by visually challenged persons with their touch sensory modality. It requires a good tactile accuracy, transforming the raised dots in the Braille to a understandable information. Expert Braille readers uses both hands for their reading purposes. They learn to move their index finger across the raised dots to identify the letters .For identifying things and locomotion , visually challenged persons uses touch in great manner.

There are evidence that plastic changes occurred within parietal somatosensory cortex, in the visually challenged persons who uses Braille reading.

No difference was reported by Pascual-Leone and Torres[4] in sensory thresholds between fifteen proficient blind Braille readers and fifteen sighted volunteers with no Braille reading ability in response to electrical, touch (von Frey hairs) and two-point stimulation.

They also reported that the area for the reading finger in the somatosensory cortex is larger compared to the contra lateral area of the same finger.[4] Thus the expanded area help them to more quickly identifying the letters in braille.

In the discrimination grating task, threshold is same for the visually challenged and for normal people by means of passive touch. The lower threshold for visually challenged persons are seen in a Braille-like discrimination task, compared to normal persons that become comparable to normal persons after behavioural training.[5] It favours that visually deprived persons uses the touch information in a efficient manner which help them for coping with their surroundings.

Neuro imaging and Braille reading:

The activation of occipital cortex to non visual function such as Braille reading is demonstrated by Sadato et al.[6] Bilateral activation of striate and extra striate cortex to Braille reading and in other touch discriminating works. A blind lady who used braille become alexic for it following both occipital cortical infarct.[7]

It is demonstrated that unimodal areas become multimodal when they are deprived of sensory function.

Ryugo et al[8] found that after de afferentation of one modality shortly after birth, dendritic spine density may increase within the modality-specific brain area of an intact modality.

Rauschecker et al[9], demonstrated expansion of cortical area of an retained sensation following a input loss in other modality.

The brain's capacity to reorganize its functions has been shown in several studies using, for example, techniques of neuro- anatomical manipulation or sensory training.[10]

Brain areas not receiving normal sensory input, owing to unmodal deprivation with an early onset, do not degenerate or remain inactive. In humans blinded at an early age, the macro anatomy of the visual cortex may appear normal in magnetic resonance images.[11]

In addition, their visual cortex has a high regional cerebral blood flow [12] and metabolic rate[11] compared with that in blindfolded sighted humans.

However, these signs of neural activation could not be related to any sensory-specific functions in these studies.

Thus, it would be intuitive to think that sensory-motor systems would be a likely locus of neural re-organization in an individual learning to read Braille.

Animal studies

Indeed, evidence from both animal and human studies have shown that tactile experience can modify the somatosensory representation of a body part such as the hand and its repeated use can lead to the enlargement of its cortical representation. [13,14]

In animal studies after visual deprivation showed the number of parietal and occipital neurons activated during tactile exploration and, further, that only very few neurons in these areas respond to visual stimuli after the end of deprivation.

The neuronal responsiveness in the occipital[15] and parietal[16] cortices after visual deprivation has been studied with multiple-cell recordings in monkeys. In the cat, anterior sylvian visual area, which normally responds to visual stimulation, becomes responsive to auditory and somatic stimulation after visual deprivation.[9] In line with these animal studies, enhanced negative d.c. potentials related to a tactile-motor task have been found over the occipital scalp in early blind humans.[17] It was subsequently shown that Braille training for 8 weeks increased tactile spatial acuity and decreased disability in dystonia patients.[18]

Polymodal association areas

Converging evidence indicates that poly modal association areas become reorganized following sensory deprivation. For example, after visual deprivation in juvenile rats, cats and monkeys, there is an increase in the number of neurons that respond to somatosensory and auditory information in multimodal areas,

including the superior colliculus, the anterior ectosylvian region in cats and the parietal cortex in primates.[19-22]

Blind subjects also have better two-point tactile discrimination skills and superior auditory recognition memory than do sighted subjects.[23]

Evoked Potential

An Evoked potential is an electrical response that follows stimulation of the CNS by a specific stimulus of the visual, auditory, or sensory system.

The external stimulai set up a discrete volley in the afferent pathways, which are recorded in the scalp with the recording electrodes.[24]

The sensory system involved and the sequence of activation of different neural structures determine the timing and location of these signals.

Chiappa et al., 1987 defined an evoked potential as the record of the electrical activity produced by groups of neurons within the spinal cord, brain stem, thalamus or cerebral hemispheres following stimulation of one or another specific system by means of visual, auditory, or somatosensory input.[25]

In a Event Related Potential study conducted in visually challenged persons and in normal subjects, revealed that posterior cerebral areas recruited in response to somatosensory stimulus discrimination. Several compensatory changes are happening in other sensory modalities, who are become visually challenged early in their life.[26]

In the clinical setting, evoked potential studies are properly an extension of the neurological examination. Studies regarding the normal somatosensory evoked potentials and brainstem Evoked Potentials are lacking in visually challenged persons.

Historical background

Historians credit Richard caton with first describing both spontaneous and evoked potentials from the brain of animals in 1875(brazier MAB 1961). The recognition by caton of this

phenomenon and its significance was amazing in a period predating the electronic amplification made possible by the vacuum tube.

The study of human CNS evoked potentials began 1942 with George Dewson's use of photographic superimposition of the post stimulus EEG in the demonstration a wave form following peripheral nerve stimulation.

He described two patients in whom sensory stimulation would provoke myoclonic seizures. During the seizure which followed a stimulus such as a tap on the tendon, the discharge in the EEG seemed to be larger on the side of the head opposite to the side stimulated. These seizures were preceded by electrical disturbance which were detectable in the scalp and whose characters suggested that they were of cerebral origin. The stimulai elicited potentials of less than several microvolts could not be appreciated as distinct from the artefact.(non event related potentials). Dawson first used a technique of photographic over trace to suppress the non event related potentials and there by allow visualisation of the time locked event potentials.

Dawson original system was unable to resolve the high frequency components of SEPs due to slow sampling rate. Dawson (1954) was structured a advanced mecheno-electrical (analog) device averaging brain potentials triggered by stimulai. Averaging was indispensable to show the event related activity, which is normally invisible in the on going background EEG activity. With the development of the signal averaging techniques it become possible to analog to individual components of the normal waveform, and this capability was better extended to delineate the characters of these discharges and the sequences of short latency potentials generated in the brachial plexus,spinal cord, and subcortical structures.

Though SEPs are in yogue for more than fifty years the clinical application of SEP were limited earlier, because of the uncertainty concerning the generator sources of the various waveforms and a lack of standardization of the recording techniques (for instance the use of multiple recording like cephalic v/s non cephalic reference are used by different investigators) . In the clinical scenario usage of SEPs are decreased due the advanced neuro radiological imaging. By assessing the conduction in the somatosensory pathways in the spinal cord and intra cranial segments SEPs have a greater clinical impact.

Somatosensory Evoked Potentials (SEP)

Somatosensory Evoked Potentials are electrical signals generated by sequential activation of specific structures along the somatosensory pathways following stimulation of peripheral nerves. The dorsal column lemniscal system is the major anatomical substrate of the SEPs within the central nervous system.

Physiology SEPs

When a mixed peripheral nerve with both sensory and motor components is stimulated both group I A muscle afferents and group II cutaneous afferents contributes to the resulting SEP.

The first order neurons are located in the dorsal root ganglia. The impulses ascend via the medial division of the root and the fasciculus gracilis and cuneatus in the dorsal column of spinal cord. The second order neurons arise from the nucleus of gracile and cuneatus and cross to the opposite side and ascend in the medial lemniscus to the ventropostero lateral nucleus of thalamus. The thalamo parietal radiations enter the posterior limb of internal capsule and terminate in the primary sensory cortex.

The SSEP determines the functional integrity of the dorsal column-medial-lemniscal system.

SEP wave forms are altered by diseases processes that impair the function of these structures as that produces conducting delays or blocks in the neural pathways connecting these. They can be elicited by virtually any sensory stimulai, such as touch or temperature changes. But the best method to obtain recordings by means of giving minute electrical stimulation repeatedly in the peripheral nerves. This produces the more consistent and reproducible waves, and stimulai can be controlled easily.

The short latency potentials occur within the first 60 msec after the electrical stimulation and is used in the clinical practice commonly and the middle and long latency potentials show a wider range wave variability makes their clinical use more difficult. They are widely used diagnostically to demonstrate the existence and often suggest location of the neurological leisons.

In addition to diagnostic testing, SEPs are increasingly being used to monitor neurological function during surgery with the goal of detecting potentially reversible neurological insults.

Nerve action potentials that travel along nerves or fiber tracts are called travelling waves. The potentials that remain located in area of the nuclei or synapse are called stationary waves.

Evoked potentials (EP) signals which comprise of both travelling and stationary waves are generated by both cortical and subcortical grey structure as well as white matter pathways.

Feinsod et al [27] demonstrated that somatosensory Event Related Potential (ERP) components have shortened peak latencies in blind humans.

Naming of components

Most investigator and the guidelines give a polarity and latency designation as the name of a component (example N 20). The names are derived from the polarity(N for negative and P for positive) followed by an integer indicating the anticipated latency in milliseconds for average sized healthy adults.

Generators of median SEPs

N9 potential arises from distal brachial plexus.

N13 has been postulated to originate from the caudal medulla and cerebellum because N13 was normally recordable in the patients after thalamic , midbrain and pontine strokes.[45] Trans oesophageal recordings of N 13 revealed the highest amplitude was present between C 3 and C 7 vertebral levels and it was not recordable above C 2 vertebra. The origin of N 13 there ore postulated below foramen magnum.[46]

The P 14 waveform persists in thalamic leison and hemi section of high cervical spinal cord suggesting that is generator lies between thalamus and lower medulla.[47-49]

These studies have removed the earlier notion about common origin of N 13 and P 14.

N 18 is probably a far field potential arising from thalamus or collateral branches in rostral brainstem.[43]

N20 potential thought to be generated from primary somatosensory cortex in the posterior lip of central fissure.[43] This however is not agreed by other authorities that considered N 20 originate from ventro postero lateral nucleus of thalamus. The later

postulation was based on the observation of absence of N 20 in patients with thalamic haemorrhage and the former on the absence of N20 in the patients with anoxic brain damage where thalamus was sspared and the cortex is necrotic.[50]

Stimulus parameters

SEP s are usually evoked by bipolar transcutaneous electrical stimulation applied on the skin over the trajectory of peripheral nerves. SEPs can be elicited from almost any nerve. The stimulation sites typically used for clinical diagnostic SEP studies are the median nerve at the wrist, and posterior tibial nerve at the ankle. Recording electrodes are placed over the scalp, the spine, and peripheral nerves proximal to the stimulation site.

Three types of stimulation and recording of SEPs:

- Mixed nerve
- Cutaneous nerve
- Dermatomal SEPs

Mixed nerve stimulation

Advantage:

Maximum amplitude, better potentials as larger diameter fibers are greater here than in cutaneous (digital) nerves.

Cutaneous nerve stimulation

Cutaneous nerve stimulation such as the sural, superficial peroneal, and lateral femoral cutaneous nerves in the lower extremity and the digital, superficial radial, and other nerves can elicit scalp SEPs. However, the amplitude of the potentials is much smaller than those obtained with mixed nerve stimulation, and potentials are not obtained over the spine.

Dermatomal

Stimulates particular dermatome, because most of the nerves are composed of 2 or 3 roots; hence this is better for segmental localisation. Stimulator is kept at the center of the dermatome so that overlap will not be there. Stimulation sites are the thumb (C6), adjacent sides of index and middle fingers (C7), little finger (C8), the dorsal surface of the foot between the first and second toes(L5) and the lateral side of the foot (S1).

Disadvantage:

It is difficult to record and the amplitude of potentials is small.

Central conduction time

The interval between N13 peak in the cervical-scalp lead and the cortical N20 peak, referred to as 'central conduction time' (CCT) (Jones, 1977; Hume and Cant, 1978) is widely used as an easy method of estimating the somatosensory conduction within the central nervous system.

This can be onset CCT and peak CCT, onset CCT correlates with height, where as peak CCT does not.

Brainstem Auditory Evoked Potentials (BAEP)

Brainstem auditory evoked potentials (BAEPs) are signals generated in the auditory nerve and brainstem after an acoustic stimulus. The other names are far field electrochleography, Auditory Brainstem response and Brain Stem audiometry.

Auditory evoked potentials have been divided into short-latency components, with latencies of under 10 msec in adults; long-latency AEPs, with latencies exceeding 50 msec; and middle-latency AEPs, with intermediate latencies.[28]

Short latency Brain stem Auditory Evoked Potentials are the clinically important in localising the lesions. They are the wave forms which are consistent and reproducible in the clinical setting than the middle and late responses.

Stimulating and recording technique

Patients will be relaxed and sitting comfortably or lying in order to reduce the myogenic artefacts. Patients are awake during the study period.

Brief acoustic clicks are delivered through headphones at a rate of 10 Hz. The stimulus intensity of 60 to 65 dB is a typical level. The contra lateral ear is masked with continuous white noise at an intensity 30 -40 dB less than the stimulating the ear intensity to prevent the waves arising from the non stimulated ear.

Placement of the recording electrodes are at the vertex Cz of the international 10 -20 system and at the both ear lobes (Ai or Ac) or at the mastoids(Mi or Mc). The ground electrodes are placed in the forehead. For optimal results same type of electrodes should be used.

2000 epoches per trial is typically used. More averaging is needed when signal to noise ratio is poor. Averages should be two separately recorded and superimposed. This is for the reproducibility.

Waveform identification and generators

The BAEP waveform typically begins with an electrical stimulus artifact that is synchronous with stimulus production at the transducer.

Roman numerals are given to the each upward peak recording as established by Jewett and Williston. The downward pointing peak are added suffix N, to the peak that follow.

Wave I is the first major up going peak of the recordings. A bifid peak is occasionally present. It arises from the first volley of action potentials in the auditory nerve at the most distal portion of the nerve.

The origin of wave I in the most distal portion of the auditory nerve is demonstrated by its presence in some patients who fulfilled the clinical and electroencephalographic criteria for brain death.[29-31] Wave I is present in at substantial amplitude in the Cz-Ac channel.

The next upward pointing peak is wave II. It may be small in certain normal subjects. It is likely that activity within the proximal end of the auditory nerve and postsynaptic activity within the cochlear nucleus both contribute to wave II. Because the auditory nerve terminals are within the substance of the cochlear nucleus, the distinction between a proximal auditory nerve generator and a cochlear nucleus generator does not have a major impact on the anatomic localization of the cause of a wave II abnormality. Wave II N, in the BAEP waveform in the same manner as the N l volley gives rise to wave I N.

The next upward peak is wave III. A bifid wave III is occasionally observed as a normal variant. The wave III latency in such waveforms can be scored as midway between the peak latencies of the two subcomponents. Superior olivary complexes or their outflow within the lateral lemniscus are the source of wave III.

Fusion of Wave IV and Wave V produce a complex morphology various from subject to subject. The IV / V complex is often the most prominent component in the BAEP wave form. This is usually followed by a prolonged large negative deflection. The identity of wave V can be clarified by changes in recording montage

or click polarity or by reducing the stimulus intensity. Consistent with generation of wave V at the level of the mesencephalon, either from the inferior colliculus itself or, as some authors have suggested.[32,33] From the fibers in the rostral portion of the lateral lemniscus as they terminate in the inferior colliculus. Intracranial data and other available recordings suggest that the mesencephalon contralateral to the stimulated ear is the major generator of wave V. the larger downward deflection following wave V predominantly reflects post synaptic potentials with in brainstem auditory nuclei, primarily the inferior colliculus.

The following upward peaks wave VI, VII is inconsistent in subjects.

Influencing factors

Males have longer latencies than females. As the age advances the latencies are increasing. Decreasing brain temperature leads to increasing latencies. Reaching adult value by two years of age. It is prolonged in neonatal period.

Minor alterations are produced during the anesthesia ,so they are used for intra operative monitoring of the ears and auditory pathways.[34,35]

Clinical interpretation of BAEPs

Waves II, IV, VI, and VII are sometimes not identifiable in normal individuals, and their peak latencies display more inter individual variability than the other waves. Amplitude measurements of the individual components are also highly variable across subjects. Therefore, clinical interpretation of a patient's BAEPs is as follows

1) Presence or absence of waves I, III, and V
2) Measurements of the wave I latency
3) I-V inter peak interval
4) I-III and III-V inter peak intervals
5) Right-left differences of these values
6) IV / V: I amplitude ratios.

Abnormalities of wave I usually reflects peripheral auditory dysfunction, either conductive or cochlear.

Lengthening of inter peak latency of I – III reflects abnormalities in the ipsilateral distal portion of eighth nerve and in the lower pons.

Abnormality of the I-III inter peak interval is the characteristic BAEP finding in eighth nerve lesions such as acoustic neuromas.

Prolongation of both the I-V and III-V inter peak intervals, or complete absence of the IV / V complex in the presence of a wave III, shows the pathology in hearing pathway between lower pons and the mesencephalon. Abnormalities in the III-V inter peak interval are seen in a variety of disease processes involving the brainstem, including demyelination, tumor, and vascular disease.

When the normal I – V latency occurs, the significance of prolongation of inter peak latencies of I – III or III – V is less clear.

Advantages and uses

The need for patient cooperation less compared to SEPs and can be used to prognosticate in comatose patients. Bilateral brainstem damage leads to absent wave forms.

MATERIALS AND METHODS

The study was conducted on the patient attenders in neurology services at the institute of neurology, Madras Medical College and Rajiv Gandhi Government general Hospital from May 2011 to march 2013. Institutional ethical committee is applied and the committee approved the study.

Informed consent was obtained from the participants. In the control group a volunteer read the information sheet and consent form are visually challenged persons are got by means of thumb impression. Visually challenged persons are chosen from a government blind school.

Twenty five persons from school and twenty five persons of age matched controls are chosen.

Inclusion and Exclusion criteria

Those who are visually challenged since childhood are selected

Persons with other associated diseases are excluded.

Participants age, sex, visual acuity and height are measured and documented.

Methods

Patients are advised to avoid applying oil after head bath on the day of test. His test is done in the sitting position with comfortable neck muscle relaxation or in the lying posture according to the patients relaxation.

Somatosensory Evoked Potentials

Machine settings

The SEPs are best recorded by amplification of the potentials between 10000-5,00,000. The impedance was kept below 5 Ω . The filler settings was 20-30 Hz for low filters and 3000 Hz for high filters. Analysis time base should be 50-6- ms which can be extended to 60-100 ms. In the latter low filter should be set at 1-3 Hz and 1000-2000 responses should be averaged . The skin was cleaned with alcohol to reduce artifacts. For median SEPs the stimulus was applied 2 cm proximal to skin crease.

Stimulus factors

A 200 microvolt square wave pulse sufficient to produce a painless twitch of the thumb in median SEPs was applied. A current ranging in amplitude from 5-15 mv was used for stimulation. The rate of stimulation was between 3hz and 8Hz. High rate stimulation is painful and result in progressive loss of amplitude and decrease in latency. Since the SEP waveforms are very amall , 1000 or more epochs were averaged.

Median SEPs

Position of electrodes:

1 cm disc electrodes filled with appropriate conducting jelly or paste was used. Stimulation is given to the wrist with the cathode 2 cm proximal to the skin crease . The electrode were placed in the following positions

1. Erb's point: the electrodes were placed 2 -3 cm above the midpoint of clavicle
2. Spinous process of the fifth cervical vertebra
3. 2 cm posterior to C3 or C4 of 10-20 international system of EEG electrode placement.

The spinal electrode is designated as c5S or C5 Sp and Erb's point electrodes EP1 and EP2 were assigned to left and right side. The montage for the median SEP s is as follows

Channel 1: C 3 / 4 – Fz

Channel 2: EP1 /EP2 – Fz

Channel 3: C5Spp –Fz

Channel 4:antecubital fossa

Median SEPs wave form

Erb's potential (N9) is seen as a negative peak in EP-z channel. Spinal potential (N 13) is a negative peak seen in C5Sp-Fz . The N20 waveform is also a negative peak seen in C 3/4 –Fz channel and is followed by a positive cortical potential P25.

The waveforms are analysed for the following parameters:

1. Latency
2. Amplitude
3. Inter peak latency

The latency is measured from the stimulus artifact to the peak of waveform and the inter peak latencies are calculated .

1. Brachial plexus to the spinal cord (N9-N13)
2. Central sensory conduction time (N13-N20)

Amplitude of wave forms are less important in clinical practice since their values are variable and do not follow a normal distribution.

Lower limb SEPs and ulnar SEP s are not done.

Brainsem Auditoru Evoked Potentials (BAEP)

Patients are awake and relaxed comfortably sitting in a chair. The auditory stimulus used is a click delivered through headphone. filter band 100-3000 hz. Each ear is stimulated with 60-70 dB above click perception with masking of the contralateral ear with intensity less than 30-40dB less than click stimulus . First 10 ms waves are recorded with average 2000 stimulai , twice the test is done and are superimposed for their consistency.

Channel and montage

Channel 1 is ipsilateral ear mastoid to vertex .

The recording electrodes are located, one for each ear lobule and a third electrode is located on the scalp at the vertex at Cz position of 10-20 international system.

The wave forms analysed:

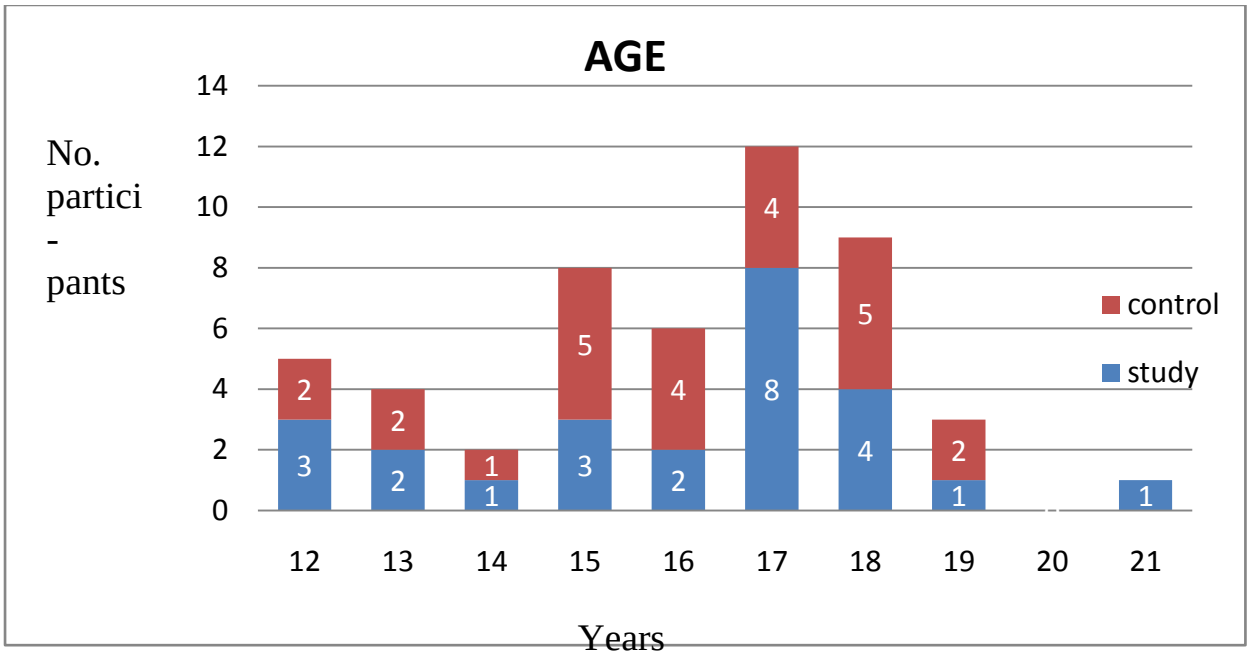
1. Latency of waves I to V
2. Inter peak latency of I – III, I -- V ,III – V

RESULTS

Twenty five visually challenged persons and the equal number of controls are participated in the study.

Age group

The age group between 12 to 21 years are participated.

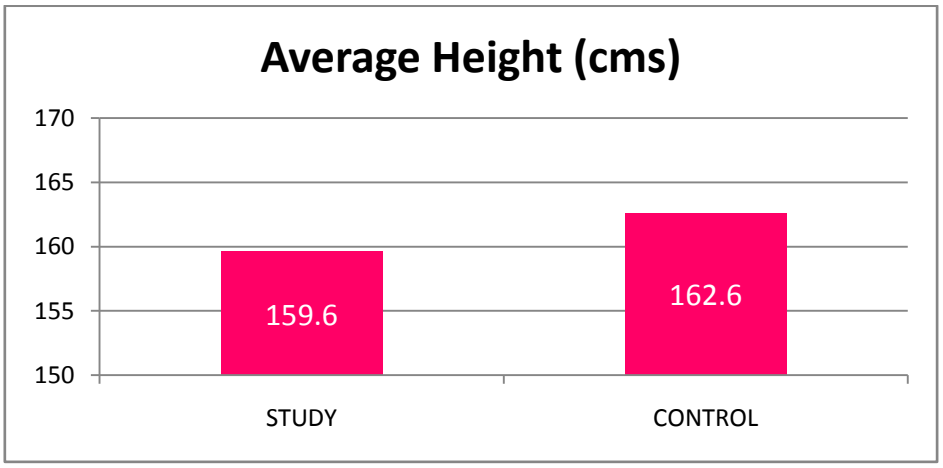


Sex

All are male in the study and control population.

Height

Average height in the study group is 159.2 cms, in the control group 162.2 cms.

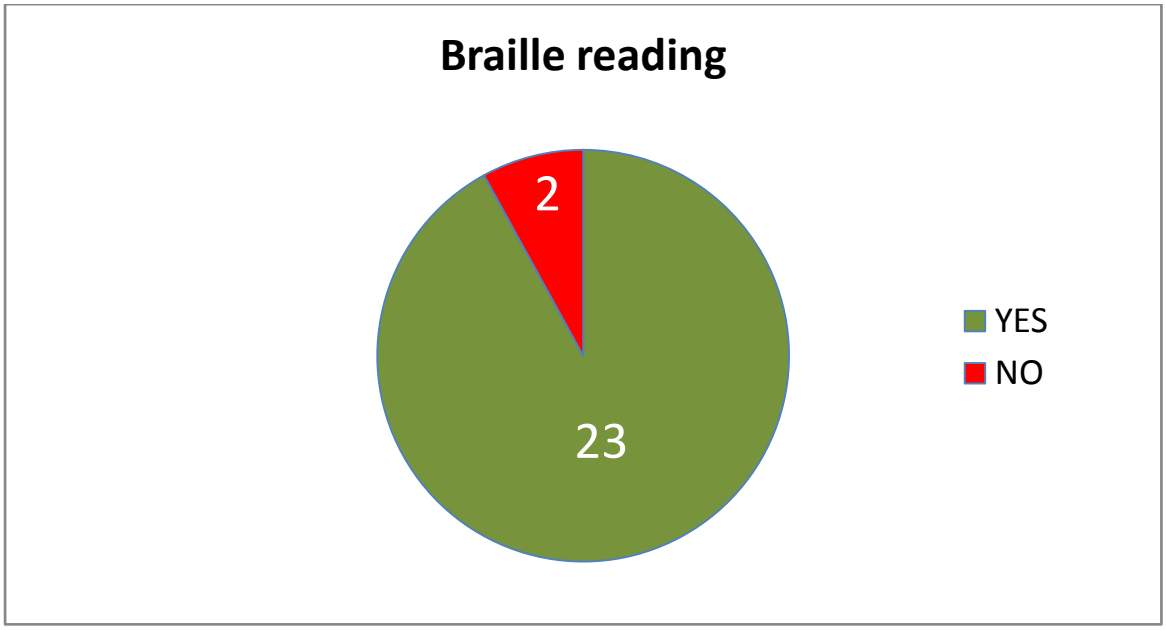


Visual acuity

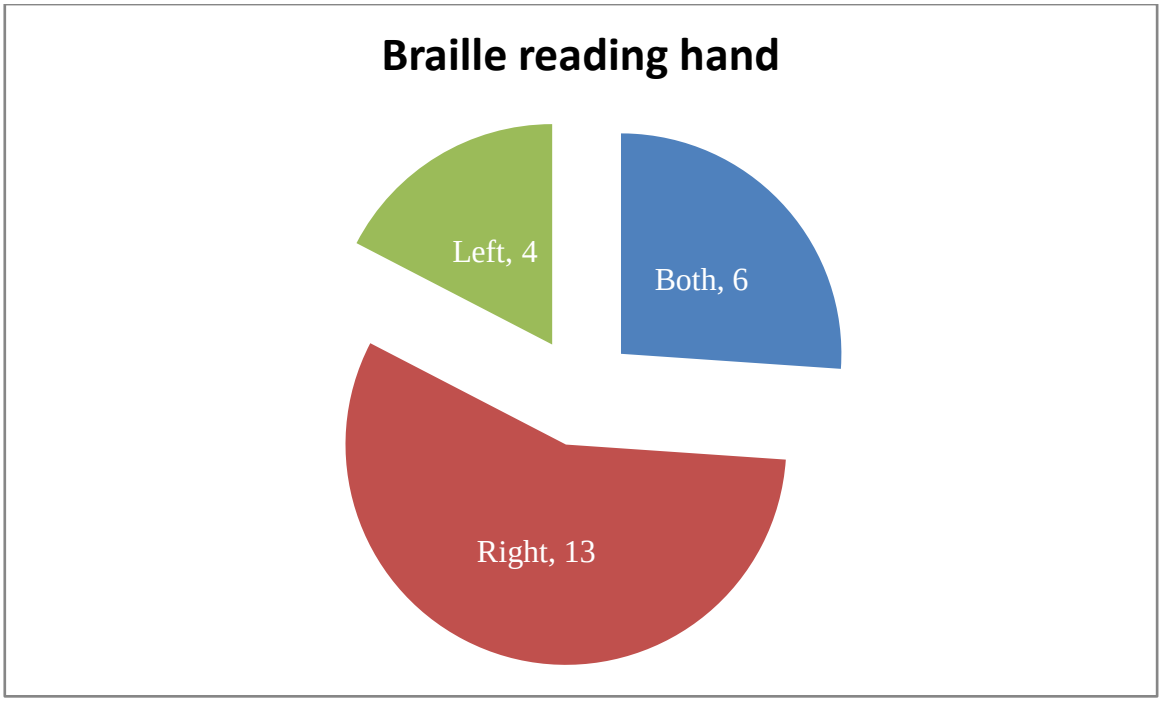
All the study population are legally blind according to the indian definition except one, who has visual acuity of 20/70 in the better eye to the 20/100 in the other eye. He can put his signature as well. He is using braille also for the 4 years.

Braille reading

Only two of the participants are not using braille in the study group.

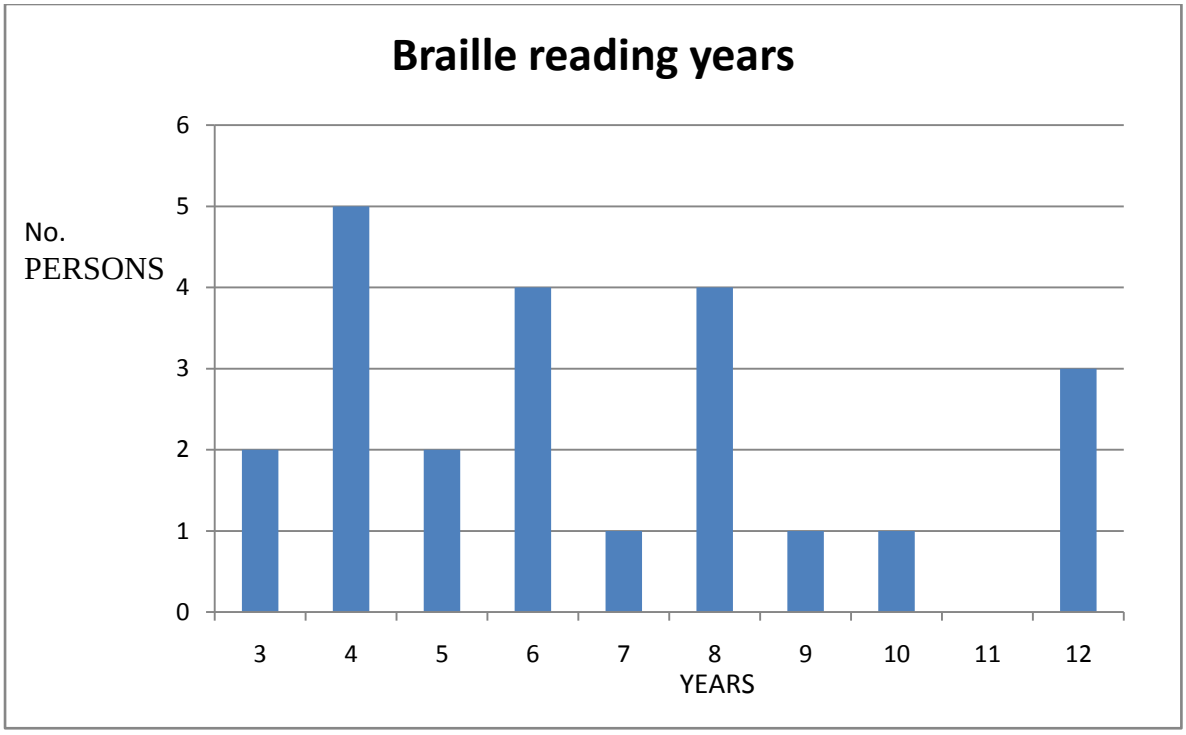


For braille reading both hands are used by 6 among 23. Right hand is used by 13 and the remaining are using the left hand.



Braille reading years

The average year of braille reading in the study population is 6.69 years, ranging from 3 years to 12 years.

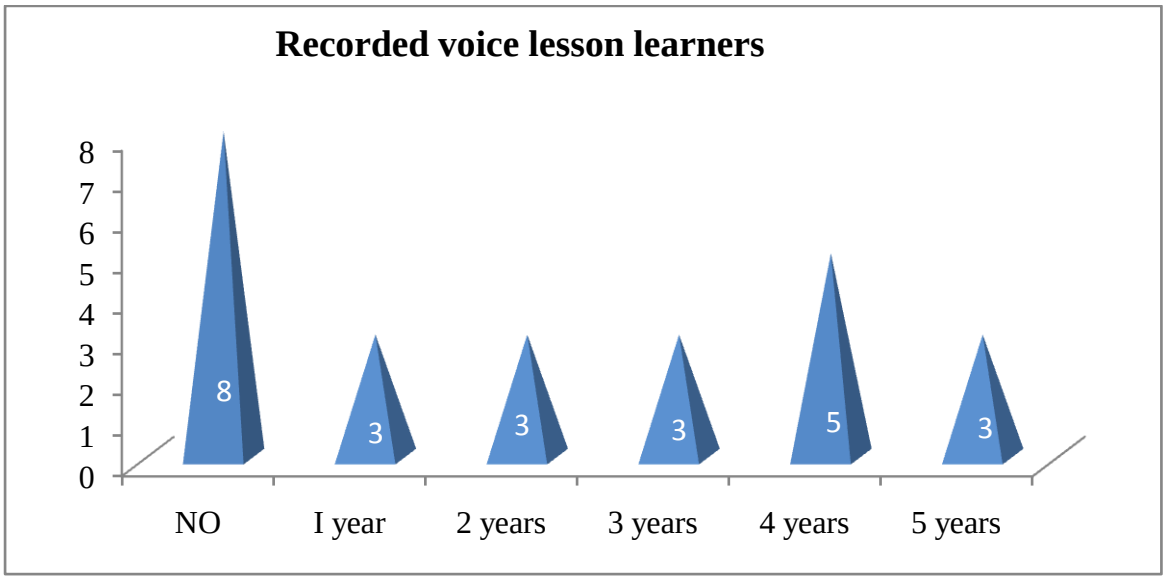


Five students are using braille for 9 to 12 years.

Auditory lesson learning:

Recorded voice lesson learning is the other method used by the visually challenged persons. They use auditory learning after 7th standard. One of the 12th standard student stopped braille reading after he completed seventh standard. Now he is using auditory learning for past 5 years. They are decreasing the braille use because of the available material for their study is scarce and the easy method of making of recorded voice from books. Only very few of visually challenged persons uses both equally.

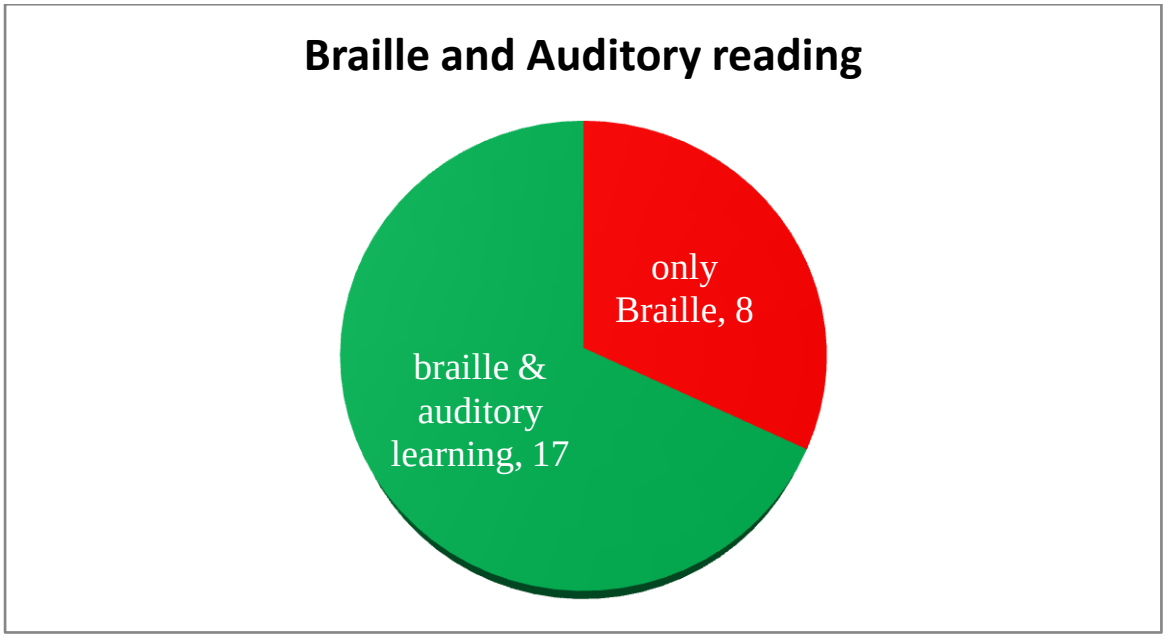
Number of years of auditory lesson learning



Auditory learning was utilised by 3 persons for 1 year, 2 years, 3years and 5 years each. 5 persons had used auditory learning for 4 years and 8 did not used auditory learning.

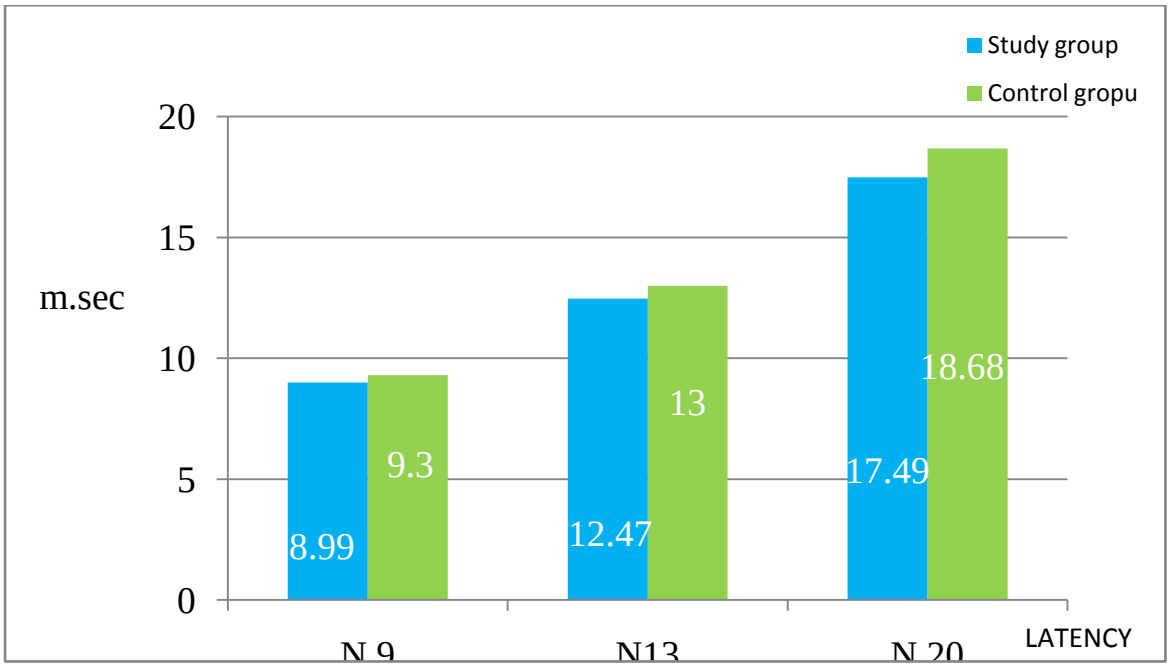
Braille reading and auditory lesson listening:

Among 25 students 8 are using braille reading as the only method of learning. Remaining 17 using auditory listening for their learning for past one to five years.



Somatosensory evoked potentials

The average values of the N 9, N 13, N20 wave latencies in the study and the control population are given below.



Wave	Study	Control	P value	Significant
N9	8.97 ± 0.71	9.32 ± 0.72	0.016	Not Significant
N13	12.47 ± 0.84	13.90 ± 0.66	< 0.001	Significant
N20	17.49 ± 1.15	18.67 ± 0.75	< 0.001	Significant

There is a significant P value for the study and control group in the N 13 and N20 waves. The latency of N 20 potentials occurs earlier in the visually challenged persons compared to the normal group.

Age and sub group results

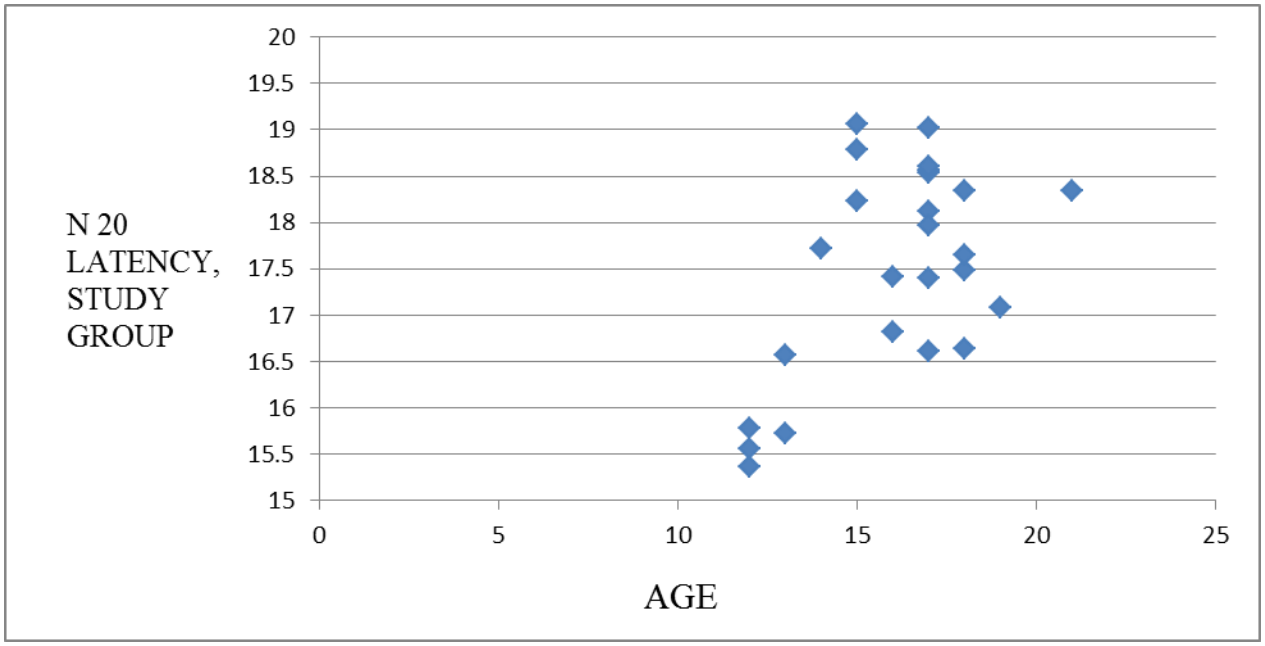
While studying the results of latencies in 12- 16 years age group, the following results are obtained.

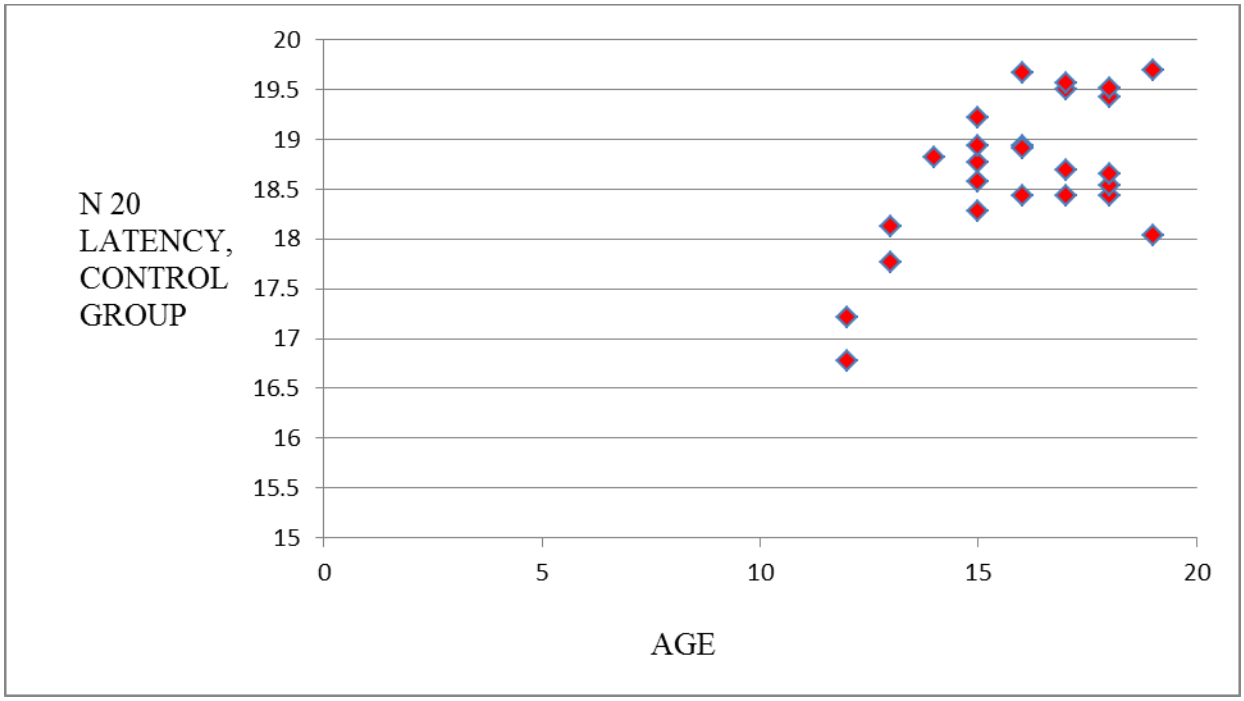
12-16 years of age	Study group	Control group	P value	Significance
	Mean ± S.D	Mean ± S.D		
N 9	8.68 ± 0.80	9.17 ± 0.85	0.055	Significant
N 13	12.22 ± 0.98	12.86 ± 0.68	0.013	Significant
N 20	17.00 ± 1.33	18.29 ± 0.78	<0.001	Significant

In the younger age 12 – 16 years , the central conduction is faster in visually challenged persons than the control group.

The average height in the 12-16 years group is 152.45 cms and 155.18 cms in the study and control population respectively.

Age and N 20 latency



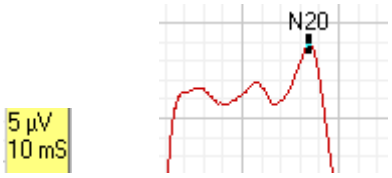


This scattered diagram shows the earlier occurrence of N20 latencies in the study group when compared to control group.

The following SEP cortical recording from the youngest boy in our study group age 12 years using Braille for 3 years.

right side

This clearly shows the N20 latency of 15.21 m sec in right side . Another cortical recoring from the study group, 13 years who decreased the braille reading and well using the recorded voice lesson learning.

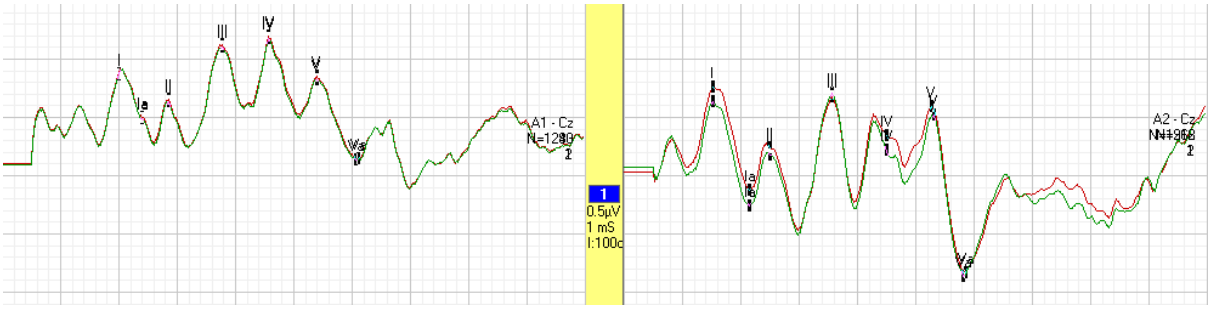


right side

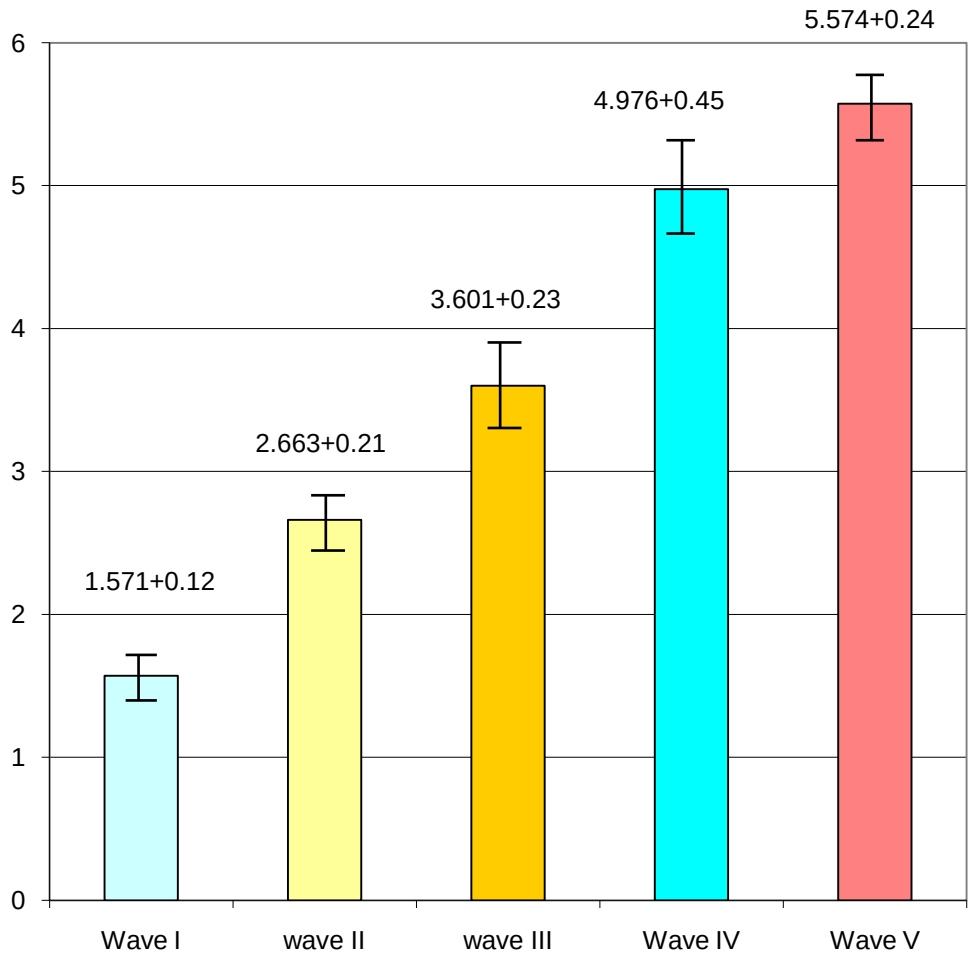
This SEP recording shows the occurrence N20 wave 16.88 m sec which is prolonged when compared the above recording.

BAEP:

The following trace shows the twice averaged, superimposed BAEP waves in a study population participant.

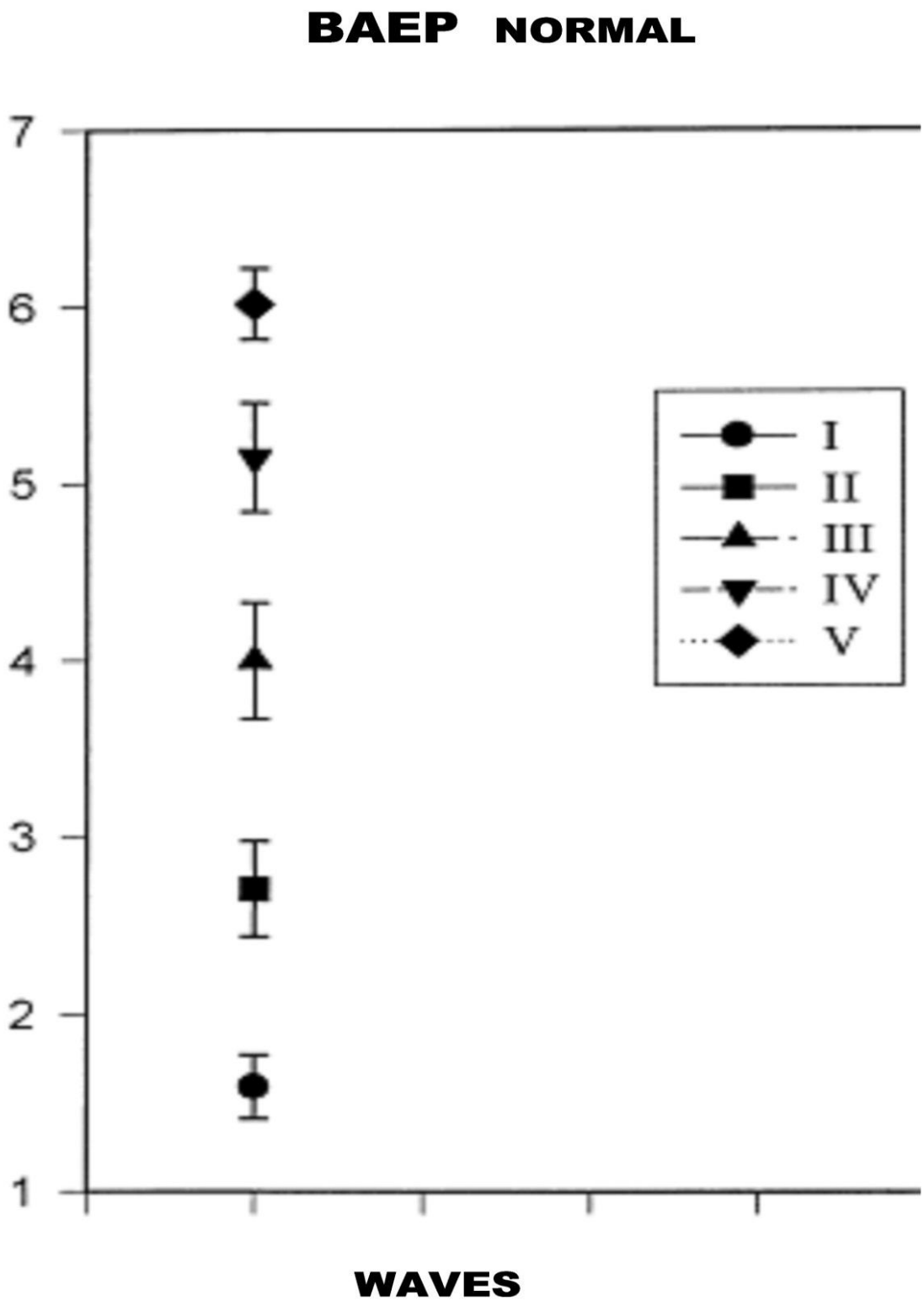


BAEP - STUDY POPULATION (MEAN ± SD)



Wave IV is not consistently present in our recordings of 50 patients.

BAEP in control population



Y axis shows the latency in m sec.

Nerve Conduction Study

Nerve conduction study of upper and lower limbs is done. The motor & sensory conduction latency, amplitude, area and nerve conduction velocity are within normal limits.

DISCUSSION

Historically, Richard Caton (1875) discovered evoked potentials and the electroencephalogram (EEG) at the same time. The evoked potentials provided a useful tool for neurophysiological research.[36] Since then evoked potentials are placed in the important diagnostic tool in neurology. Our patients somatosensory evoked response values are in comparison with available studies.

Chiappa et al , recorded median mean absolute latency of N 19 wave was 19.0 with standard deviation of 1.02. Minimum latency of N 19 wave was 16.7 m sec and the maximum was 21.2 msec.

In our study, N 20 mean value is 18.67 m sec with a standard deviation of 0.75 in the controls with range from 16.78 m sec to 19.70 m sec. In the visually challenged persons with a mean value of N 20 17.4 m sec , ranges from 15.37 m sec to 19.06 m sec. Peak latency of N 20 wave occurs earlier in the visually challenged persons using Braille when compared with control population.

In another study by Jones et al[37] with 33 arms stimulated in 22 normal subjects in the age group from 19 to 33 with mean 24 years

found that the N 9 latency was 8.7± 0.5 msec . N 13 latency recorded in the C 7 vertebra level showed the value of 12.7±0.4 m sec. Scalp recorded mean N 20 latency with stimulation at wrist was 18.3 m sec. Our study group mean N 20 value 18.67 m sec , which is comparable with it. But the age group in study group is from 12 to 21 years.
Arm length was not mentioned in their study.

N 13 values in the control group is 13.90 ± 0.66 m sec. which is prolonged when compared to their study. But in his study the N 13 recording was done in the C 7 vertebra, but we used C 5 vertebra recording. He also said the onset and peak latencies of the spinal potentials and N 20 are correlate with height but not age.

Inter peak latency

Inter peak latency	Mean m sec		
	Chiappa et al	Our study	
		Control	Study
N9 to N 13	3.8	3.78	3.49
N20 to N13	5.5	5.58	5.02
N20-N9	9.3	9.35	8.52

The mean N 9 to N 20 inter peak latency is 8.56 m sec. The conduction time is faster in the visually challenged persons using braille than in control which is 9.35 m sec.

Minimum and maximum inter peak latency:

Inter peak latency	Min m sec			Max m sec		
	Chiappa et al	Our study		Chiappa et al	Our study	
		Control	Study		Control	Study
N9 to N 13	2.7	3.16	2.30	4.5	4.56	4.26
N20 to N13	4.7	4.90	3.46	6.8	6.49	6.42
N20-N9	7.8	8.21	7.33	10.4	10.85	9.88

In our study the minimum and maximum values of inter peak latencies are similar to Chiapp et al. the inter peak latencies also short in visually challenged persons , indicating fast conduction than controls.

Central conduction time(CCT)

Peak central conduction time (or) cord cortex conduction time is the measurement of peak of N13 to peak of N 20 waves.

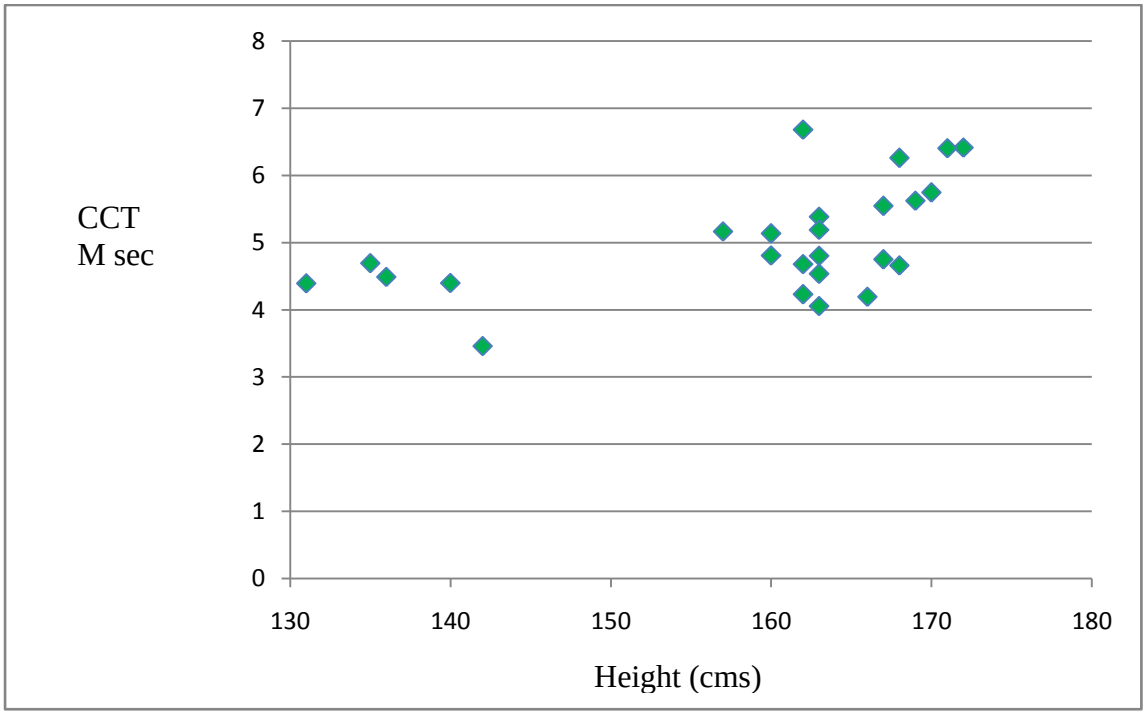
D.Zegers de Beyl analysed the central conduction time in the 40 normal subjects in the age group 48.2± 23.3 years .

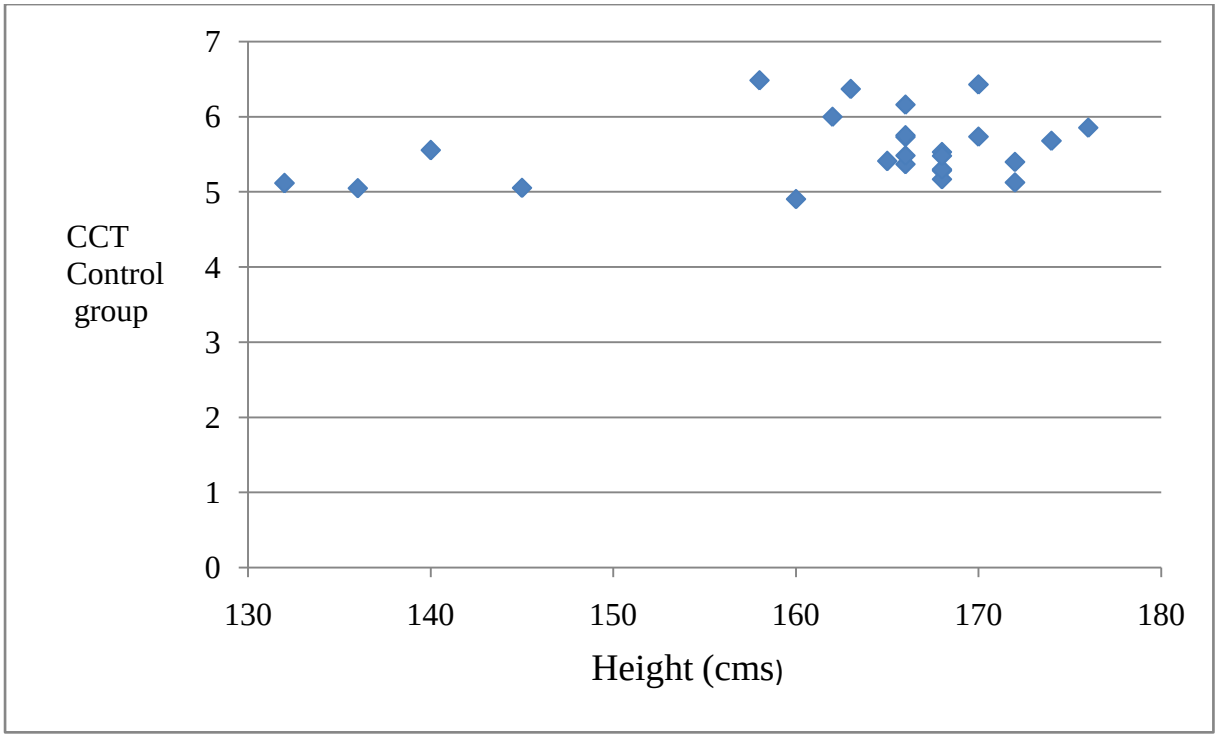
Author	Central conduction time
	Mean ± SD
D,Zegerde Beyl (1988)	5.70 ± 0.54
Rossini(1983)	5.68 ± 0.45
Yamada(1986)	5.5 ± 0.6
Chiappa	5.5
Control group	5.58
Study group	5.02

The central conduction time is short in the visually challenged group. This shows the conduction between cervical cord to cortex is faster compared to the normal population. This analysis shows the parietal cortex perceives the tactile sensations earlier in the visually challenged persons compared to the normal population.

IPL	Study	Control	P value	Significant
N9 - N13	3.467 ± 0.533	3.776 ± 0.303	0.023	Significant
N13 - N 20	5.028 ± 0.816	5.577 ± 0.444	0.005	Significant
N 9 - N20	8.496 ± 0.747	4.353 ± 0.599	< 0.001	Significant

CCT and height:





Central conduction time does not correlated with height as comparable with Ozaki et al[44] study. In he studied the SEPs in 72 normal subjects.

With mean value of peak CCT 5.7 ± 0.5 m sec, and onset CCT 6.2 ±0.4 m sec he stated there was a significant correlation between onset CCT , but not with the peak CCT and height for the median nerve SEPs. He also found that there was no correlation with right left difference to the onset or peak CCT.

All these factors strongly favours our result, faster central conduction time in the visually challenged persons using braille.

SEPs and arm length

M.J.Taylor[38] studied the normal somatosensory evoked potentials in paediatric population. His results are in the following table.

AGE	ARM LENGTH (cms)	N 20 (m sec)	UPPER LIMIT m sec
12-16 years	66	17.24± 0.69	18.97
14-18 years	73	17.73± 0.79	19.7
ADULT	74	18.38± 0.89	20.61

Our results

Age	Group	ARM LENGTH	N20 LATENCY	UPPER LIMIT
		cms	mean ± sd	m sec
12- 16 years	Study	76.2	17.0 ± 1.33	19.06
	Control	77.6	18.29 ± 0.78	19.22

The peak latencies of N 20 wave with 3 Hz and 30 Hz simulation ranges from 18.1 to 20.2 msec, 19.0 to 21.1 msec respectively in the 10 subjects studied by G.M.Manzano.[39] Their age range was from 18 years to 37 years.

Among our control population, 7 are in the age of 18 and 19 years. Their peak latency of N 20 wave ranges from 17.96 to 19.76 msec. The study population in the age group of 18 – 19 years are five in number. Their peak latency of N 20 wave range from 16.4 to 18.48 msec. They are using Braille for 8 -12 years, now with more of recorded voice learning for 4- 5 years.

IFCN value and our study

Mean Latency (msec)			
	IFCN Value	Our study population	Our control population
N9	9.8	8.99	9.32
N9-N13	3.5	3.49	3.78
N20 - N9	9.8	8.52	9.35
N20- N13	5.7	5.02	5.58

Upper limit of normal (msec)			
	IFCN Value	Our study population	Our control population
N9-N13	4.4	4.26	4.56
N20 - N9	11	9.88	10.85
N20- N13	7.2	6.42	6.49

Our SEP s results are well comparable with the IFCN[43] recommendations published in 1994.

BAEP:

Our recorded values of waves are well in normal limits.

Mean latency (mean ± SD)				
Wave	Masakazu [42]	Chiappa[40]	Our study	Our Control
I	1.60 ± 0.0815	1.7 ± 0.153	1.54 ± 0.17	1.58 ± 0.11
III	3.60 ± 0.22	3.9 ± 0.19	3.60 ± 0.23	3.62 ± 0.22
V	5.58 ± 0.25	5.7 ± 0.25	5.57 ± 0.23	5.60 ± 0.22

The absolute latency of wave V is normally less than 6.4 msec. The right-left asymmetry of the wave V absolute latency is normally 0.5 msec or less.

Inter peak latency:

Inter peak mean latency (mean ± SD)				
Wave	Chi Ran Hnang [41]	Chiappa[40]	Our study	Our Control
I - III	2.08 ± 0.11	2.1 ± 0.15	2.03 ± 0.27	2.03 ± 0.26
I - V	4.01 ± 0.18	4.0 ± 0.23	4.00 ± 0.28	4.01 ± 0.26
III - V	1.93 ± 0.15	1.2 ± 0.16	2.16 ± 0.45	2.16 ± 0.42

Our I -V inter peak latency is well comparable with available studies.

III –V latency is higher when compared to the Chiappa et al.[40] Due the inconsistency of wave IV in our study may be lead to the fusion the wave form and that may be reason the same.

CONCLUSION

1. The central conduction time is significantly short in the visually challenged persons using Braille reading. It favours that visually deprived persons uses the touch information in a efficient manner which help them for coping with their surroundings.
2. The fast conduction decreases with decreasing usage of braille reading.
3. Brainstem auditory evoked response is normal in visually challenged persons
4. Brainstem auditory evoked response not faster when they are using recorded voice lesson learning.
5. Peak central conduction time does not correlate with age.
6. In Braille reading persons , sensory nerve conduction latency, amplitude, velocities are normal while the central conduction is short indicating faster conduction.
7. The faster central conduction is of significance in visually challenged as they rely on touch for their locomotion and many other day to day activities. Hence it may be better if they continue to use Braille which helps better touch perception. Where as when they switch to auditory learning the central

conduction in somatosensory pathway slows to that of normal people, which may be a disadvantage to the visually challenged who has to ‘feel’ the world that they cannot see. So we recommend that Braille usage to be continued in older visually challenged persons even though they may also use auditory learning.

ABBREVIATIONS

SEPs – Somatosensory evoked potential

BAEP – Brainstem Auditory Evoked Potential

IFCN –International Federation of Clinical Neurophysiology

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SL. NO	NAME	AGE	STD	VISUAL ACUITY(1)				BRAILLE READING				RECORDED LESSON LEARNINGY EARS	HEIGHT (cms)
				RIGHT	LEFT	RIGHT	LEFT	YEARS	BOTH	RIGHT	LEFT		
1	GOPI	12	7	20/400	NPL	6/120	NPL	3		1		NO	131
2	TAMILMANI	12	7	20/400	PL	6/120	PL	4		1		NO	136
3	MANOKARAN	12	7	20/400	20/400	6/120	6/120	4	1			NO	135
4	PRABHAKARAN	13	8	NPL	20/400	NPL	6/120	3		1		NO	140
5	DHINAKARAN	13	8	20/70	20/100	6/24	6/30	4		1		1	142
6	PANDIAN	14	6	20/200	20/100	6/60	6/30	NO				NO	163
7	SHREE KRISHNASEKAR RAO	15	9	NPL	NPL	NPL	NPL	9		1		2	170
8	SHARUKKAN	15	8	PL	PL	PL	PL	4	1			1	163
9	S.ARUNKUMAR	15	9	20/800	NPL	PL	NPL	6		1		1	172
10	MARIMUTHU	16	11	NPL	NPL	NPL	NPL	7	1			4	162
11	S.RANGARAJ	16	11	NPL	NPL	NPL	NPL	6	1			4	163
12	ARUNRAJ	17	11	20/400	20/400	6/120	6/120	4		1		3	163
13	G.SHANMUGASUNDARAM	17	12	20/200	20/400	6/60	6/120	8	1			3	162
14	SOUNDAPPAN	17	11	NPL	NPL	NPL	NPL	8		1		3	168
15	JUSTIN	17	9	NPL	20/400	NPL	6/120	5	1			2	169
16	S.PRASANTH	17	11	NPL	NPL	NPL	NPL	10			1	NO	160
17	R.KARTHIEYAN	17	11	20/400	NPL	6/120	NPL	6		1		5	162
18	A.VENGADESH	17	9	NPL	NPL	NPL	NPL	5		1		2	171
19	ASHAN ALI	17	9	NPL	NPL	NPL	NPL	6		1		NO	160
20	MANIKANDAN	18	12	20/200	NPL	6/60	NPL	12			1	4	167
21	G.ANANDHAN	18	11	PL	PL	PL	PL	8		1		5	168
22	MANIKANDAN	18	8	PL	PL	PL	PL	8			1	NO	166
23	G.SARAVANAN	18	12	PL	PL	PL	PL	12			1	4	157
24	JEEVANANTHAM	19	12	NPL	NPL	NPL	NPL	12		1		4	163
25	S.SARANRAJ	21	12	20/200	NPL	6/60	NPL	NO				5	167

SOMATOSENSORY EVOKED POTENTIALS VISUALLY CHALLENGED POPULATION

		LEFT			RIGHT			TOTAL(RIGHT+LEFT)			TOTAL/2			INTERPEAK LATENCY		
SL.NO	AGE	N9	N13	N20	N9	N13	N20	N9	N13	N20	N9	N13	N20	N9-N13	N20-N13	N20-N9
1	12	7.71	11.02	15.52	7.71	10.92	15.21	16.71	21.94	30.73	8.355	10.97	15.365	2.615	4.395	7.01
2	12	7.8	10.97	15.74	7.85	11.17	15.38	15.65	22.14	31.12	7.825	11.07	15.56	3.245	4.49	7.735
3	12	7.69	11.17	15.82	7.83	10.99	15.73	15.52	22.16	31.55	7.76	11.08	15.775	3.32	4.695	8.015
4	13	7.8	11.39	15.69	7.84	11.26	15.76	15.64	22.65	31.45	7.82	11.325	15.725	3.505	4.4	7.905
5	13	8.96	13.42	16.25	8.75	12.79	16.88	17.71	26.21	33.13	8.855	13.105	16.565	4.25	3.46	7.71
6	14	9.17	13.31	17.65	9.35	13.06	17.79	18.52	26.37	35.44	9.26	13.185	17.72	3.925	4.535	8.46
7	15	10.21	13.21	19.58	10.1	13.42	18.54	20.31	26.63	38.12	10.155	13.315	19.06	3.16	5.745	8.905
8	15	9.17	12.58	18.33	9.38	13.12	18.14	18.55	25.7	36.47	9.275	12.85	18.235	3.575	5.385	8.96
9	15	9.23	12.26	18.67	9.48	12.47	18.89	18.71	24.73	37.56	9.355	12.365	18.78	3.01	6.415	9.425
10	16	8.96	13.12	17.08	8.75	12.05	16.55	17.71	25.17	33.63	8.855	12.585	16.815	3.73	4.23	7.96
11	16	8.38	12.86	17.94	8.84	12.35	16.88	17.22	25.21	34.82	8.61	12.605	17.41	3.995	4.805	8.8
12	17	9.58	13.28	18.33	9.79	13.42	18.75	19.37	26.7	37.08	9.685	13.35	18.54	3.665	5.19	8.855
13	17	9.73	13.63	17.92	9.5	13.26	18.33	19.23	26.89	36.25	9.615	13.445	18.125	3.83	4.68	8.51
14	17	9.29	14.17	18.16	9.39	13.63	18.96	18.68	27.8	37.12	9.34	13.9	18.56	4.56	4.66	9.22
15	17	7.58	11.63	17.5	7.46	11.92	17.3	15.04	23.55	34.8	7.52	11.775	17.4	4.255	5.625	9.88
16	17	9.17	11.42	16.71	8.75	12.17	16.5	17.92	23.59	33.21	8.96	11.795	16.605	2.835	4.81	7.645
17	17	9.76	11.97	18.71	9.48	11.88	18.5	19.24	23.85	37.21	9.62	11.925	18.605	2.305	6.68	8.985
18	17	9.28	12.48	18.98	9.17	12.76	19.07	18.45	25.24	38.05	9.225	12.62	19.025	3.395	6.405	9.8
19	17	9.11	12.82	17.9	9.2	12.84	18.03	18.31	25.66	35.93	9.155	12.83	17.965	3.675	5.135	8.81
20	18	9.3	13.18	17.37	9.48	12.29	17.6	18.78	25.47	34.97	9.39	12.735	17.485	3.345	4.75	8.095
21	18	9.24	12.28	18.2	9.28	11.88	18.48	18.52	24.16	36.68	9.26	12.08	18.34	2.82	6.26	9.08
22	18	9.38	11.96	16.4	9.24	12.92	16.87	18.62	24.88	33.27	9.31	12.44	16.635	3.13	4.195	7.325
23	18	9.12	12.28	17.58	9.32	12.69	17.72	18.44	24.97	35.3	9.22	12.485	17.65	3.265	5.165	8.43
24	19	9.17	12.38	17.08	9.17	13.67	17.08	18.34	26.05	34.16	9.17	13.025	17.08	3.855	4.055	7.91
25	21	9.17	12.16	17.92	9.58	13.42	18.75	18.75	25.58	36.67	9.375	12.79	18.335	3.415	5.545	8.96
MEAN		223.96	310.95	437.03	224.69	312.35	437.69	449.94	623.3	874.72	224.97	311.65	437.36	86.68	125.71	212.39
											8.9988	12.466	17.4944	3.4672	5.0284	8.4956

SOAMTOSENSORY EVOKED POTENTIALS IN CONTROL POPULATION

			LEFT			RIGHT			HEIGHT
SL.NO	NAME	AGE	N9	N13	N20	N9	N13	N20	(cms)
1	SARATH KUMAR	12	8.53	11.58	16.8	8.61	11.88	16.76	136
2	PRIYADHARSHAN	12	8.76	11.93	17.1	8.3	12.26	17.32	132
3	JAYAKUMAR	13	7.92	12.06	17.6	8.36	12.36	17.93	140
4	MICHEL	13	8.3	12.86	18.3	8.73	13.29	17.96	145
5	VIJAYAN	14	8.16	12.4	18.67	7.78	12.26	18.96	158
6	GOWTHAM	15	9.7	13.06	19.48	9.56	12.64	18.96	163
7	VINAYAGAMURTHY	15	10.21	13.3	18.96	10.42	13.68	18.59	168
8	BASKAR	15	9.46	13.1	18.4	9.79	13.64	18.15	160
9	PALANI KUMAR	15	9.7	13.6	18.76	9.67	13.46	19.1	172
10	MAHANDRAN	16	10.56	13.76	19.1	10.24	13.76	18.76	168
11	RAJA	16	9.3	12.8	18.76	9.76	13.24	18.1	165
12	DURAI RAJ	17	8.79	12.6	18.6	8.56	12.46	18.78	166
13	NAWZAT	17	8.4	12.26	18.3	8.96	12.6	18.56	162
14	BALAJI	17	9.3	12.86	19.3	9.56	13.28	19.7	170
15	BALASUBRAMANIYAN	17	10.37	14.06	19.76	10.1	13.6	19.36	166
16	KESAVAN	18	9.57	13.26	18.76	9.8	13.02	18.12	168
17	VISWANATH	18	10.1	13.46	19.26	9.78	13.67	19.58	176
18	BHARANI KUMAR	18	9.36	13.28	18.32	9.58	13.06	18.76	166
19	SENTHIL KUMAR	18	9.3	13.6	18.74	9.26	13.46	18.57	172
20	MANIVANNAN	18	9.62	13.7	19.28	9.31	14.28	19.76	168
21	AJITH	15	8.96	12.5	18.78	9.4	13.67	18.36	166
22	MUTHUKUMAR	16	10.28	13.78	19.56	10.1	14.06	19.78	166
23	VARUN	16	9.28	13.06	18.78	9.1	13.28	19.03	170
24	EZHILVENDHAN	19	8.38	12.48	18.1	8.56	12.62	17.96	168
25	MURTHY	19	10.28	13.78	19.56	10.26	14.26	19.84	174
			232.59	325.13	467.03	233.55	329.79	466.75	
MEAN			9.3036	13.0052	18.6812	9.342	13.1916	18.67	

SOMATOSENSORY EVOKED POTENTIALS IN CONTROL POPULATION

SL.NO	NAME	TOTAL(RIGHT+LEFT)			TOTAL/2			INTER PEAK LATECY		
		N9	N13	N20	N9	N13	N20	N9-N13	N20-N13	N20-N9
1	SARATH KUMAR	17.14	23.46	33.56	8.57	11.73	16.78	3.16	5.05	8.21
2	PRIYADHARSHAN	17.06	24.19	34.42	8.53	12.095	17.21	3.565	5.115	8.68
3	JAYAKUMAR	16.28	24.42	35.53	8.14	12.21	17.765	4.07	5.555	9.625
4	MICHEL	17.03	26.15	36.26	8.515	13.075	18.13	4.56	5.055	9.615
5	VIJAYAN	15.94	24.66	37.63	7.97	12.33	18.815	4.36	6.485	10.845
6	GOWTHAM	19.26	25.7	38.44	9.63	12.85	19.22	3.22	6.37	9.59
7	VINAYAGAMURTHY	20.63	26.98	37.55	10.315	13.49	18.775	3.175	5.285	8.46
8	BASKAR	19.25	26.74	36.55	9.625	13.37	18.275	3.745	4.905	8.65
9	PALANI KUMAR	19.37	27.06	37.86	9.685	13.53	18.93	3.845	5.4	9.245
10	MAHANDRAN	20.8	27.52	37.86	10.4	13.76	18.93	3.36	5.17	8.53
11	RAJA	19.06	26.04	36.86	9.53	13.02	18.43	3.49	5.41	8.9
12	DURAI RAJ	17.35	25.06	37.38	8.675	12.53	18.69	3.855	6.16	10.015
13	NAWZAT	17.36	24.86	36.86	8.68	12.43	18.43	3.75	6	9.75
14	BALAJI	18.86	26.14	39	9.43	13.07	19.5	3.64	6.43	10.07
15	BALASUBRAMANIYAN	20.47	27.66	39.12	10.235	13.83	19.56	3.595	5.73	9.325
16	KESAVAN	19.37	26.28	36.88	9.685	13.14	18.44	3.455	5.3	8.755
17	VISWANATH	19.88	27.13	38.84	9.94	13.565	19.42	3.625	5.855	9.48
18	BHARANI KUMAR	18.94	26.34	37.08	9.47	13.17	18.54	3.7	5.37	9.07
19	SENTHIL KUMAR	18.56	27.06	37.31	9.28	13.53	18.655	4.25	5.125	9.375
20	MANIVANNAN	18.93	27.98	39.04	9.465	13.99	19.52	4.525	5.53	10.055
21	AJITH	18.36	26.17	37.14	9.18	13.085	18.57	3.905	5.485	9.39
22	MUTHUKUMAR	20.38	27.84	39.34	10.19	13.92	19.67	3.73	5.75	9.48
23	VARUN	18.38	26.34	37.81	9.19	13.17	18.905	3.98	5.735	9.715
24	EZHILVENDHAN	16.94	25.1	36.06	8.47	12.55	18.03	4.08	5.48	9.56
25	MURTHY	20.54	28.04	39.4	10.27	14.02	19.7	3.75	5.68	9.43
					233.07	327.46	466.89	94.39	139.43	233.82
					9.3228	13.0984	18.6756	3.7756	5.5772	9.3528

BAEP IN VISUALLY CHALLENGED PERSONS

	LEFT								RIGHT							
SL.NO	I	II	III	IV	V	I-III	I-V	III-V	I	II	III	IV	V	I-III	I-V	III-V
1	1.62	2.55	3.77	4.56	5.4	2.15	3.78	1.63	1.52	2.5	3.56	4.5	5.27	2.04	3.75	2.23
2	1.46	2.42	3.68	4.48	5.54	2.22	4.08	1.86	1.52	2.48	3.76	5.2	5.76	2.24	4.24	2.72
3	1.48	2.58	3.7	5.65	5.74	2.22	4.26	2.04	1.42	2.76	3.58	4.65	5.66	2.16	4.24	2.82
4	1.44	3.08	3.94	4.67	5.54	2.5	4.1	1.6	1.71	2.96	3.71	5.68	5.75	2	4.04	2.33
5	1.44	2.94	3.35	5.46	5.6	1.91	4.16	2.25	1.52	2.52	3.6	4.67	5.21	2.08	3.69	2.17
6	1.6	2.52	3.69	5.4	6.22	2.09	4.62	2.53	1.56	2.6	3.35	4.48	5.65	1.79	4.09	2.53
7	1.44	2.19	3.44	5.24	5.78	2	4.34	2.34	1.42	3.08	3.77	5.38	5.52	2.35	4.1	2.68
8	1.6	2.9	3.85	5.16	5.27	2.25	3.67	1.42	1.48	2.69	3.94	4.82	5.6	2.46	4.12	2.64
9	1.73	2.5	3.94	4.68	5.21	2.21	3.48	1.27	1.44	2.56	3.4	4.96	5.4	1.96	3.96	2.52
10	1.77	2.94	3.48	5.1	5.81	1.71	4.04	2.33	1.78	3.08	3.44	5.74	6.15	1.66	4.37	2.59
11	1.55	2.84	3.42	4.87	5.76	1.87	4.21	2.34	1.5	2.78	3.56	5.58	5.88	2.06	4.38	2.88
12	1.41	2.6	3.74	5.39	5.46	2.33	4.05	1.72	1.54	2.66	3.56	5.43	5.62	2.02	4.08	2.54
13	1.48	3.08	3.94	4.68	5.21	2.46	3.73	1.27	1.56	2.52	3.69	4.38	5.35	2.13	3.79	2.23
14	1.6	2.3	3.35	4.28	5.58	1.75	3.98	2.23	1.72	2.58	3.88	5.1	5.21	2.16	3.49	1.77
15	1.56	3.08	3.48	4.9	5.21	1.92	3.65	1.73	1.98	2.73	3.45	4.68	5.21	1.47	3.23	1.25
16	1.62	2.56	3.66	5.34	5.42	2.04	3.8	1.76	1.54	2.8	3.52	5.54	5.66	1.98	4.12	2.58
17	1.44	2.76	3.76	4.8	5.56	2.32	4.12	1.8	1.52	2.66	3.62	4.38	5.7	2.1	4.18	2.66
18	1.42	2.73	3.85	5.2	5.46	2.43	4.04	1.61	1.9	2.5	3.48	4.56	5.21	1.58	3.31	1.41
19	1.56	2.48	3.68	4.59	5.6	2.12	4.04	1.92	1.48	2.62	3.52	5.68	5.8	2.04	4.32	2.84
20	1.64	2.58	3.56	4.64	5.4	1.92	3.76	1.84	1.56	2.66	3.72	4.4	5.66	2.16	4.1	2.54
21	1.7	2.62	3.5	5.56	5.7	1.8	4	2.2	1.62	2.68	3.4	4.52	5.56	1.78	3.94	2.32
22	1.68	2.68	3.66	5.2	5.86	1.98	4.18	2.2	1.59	2.78	2.52	4.27	5.72	0.93	4.13	2.54
23	1.54	2.54	3.7	4.78	5.66	2.16	4.12	1.96	1.62	2.46	3.54	5.38	5.4	1.92	3.78	2.16
24	1.66	2.46	3.62	5.52	5.62	1.96	3.96	2	1.58	2.62	3.76	4.58	5.76	2.18	4.18	2.6
25	1.48	2.4	3.4	4.5	5.56	1.92	4.08	2.16	1.54	2.56	3.54	5.6	5.8	2	4.26	2.72
	38.92	66.33	91.16	124.65	139.17	52.24	100.25	48.01	39.62	66.84	88.87	124.16	139.51	49.25	99.89	60.27

BAEP CONTROL GROUP

		LEFT								RIGHT							
SL.NO	AGE	I	II	III	IV	V	I-III	I-V	III-V	I	II	III	IV	V	I-III	I-V	III-V
1	12	1.64	2.44	3.68	4.42	5.52	2.04	3.88	1.84	1.56	2.54	3.64	4.58	5.34	2.08	3.78	2.22
2	12	1.49	2.46	3.74	4.52	5.54	2.25	4.05	1.8	1.54	2.52	3.78	5.24	5.78	2.24	4.24	2.7
3	13	1.48	2.58	3.72	5.42	5.72	2.24	4.24	2	1.44	2.78	3.58	4.68	5.76	2.14	4.32	2.88
4	13	1.46	3.06	3.94	4.78	5.56	2.48	4.1	1.62	1.73	2.92	3.74	5.48	5.76	2.01	4.03	2.3
5	14	1.44	2.96	3.35	5.48	5.62	1.91	4.18	2.27	1.54	2.58	3.68	4.69	5.28	2.14	3.74	2.2
6	15	1.58	2.52	3.68	5.44	6.22	2.1	4.64	2.54	1.56	2.64	3.35	4.48	5.68	1.79	4.12	2.56
7	15	1.51	2.19	3.48	5.24	5.78	1.97	4.27	2.3	1.42	3.02	3.77	5.47	5.52	2.35	4.1	2.68
8	15	1.62	2.88	3.88	5.16	5.34	2.26	3.72	1.46	1.56	2.84	3.94	4.82	5.64	2.38	4.08	2.52
9	15	1.76	2.52	3.94	4.88	5.25	2.18	3.49	1.31	1.44	2.68	3.46	4.96	5.46	2.02	4.02	2.58
10	16	1.79	2.92	3.52	5.24	5.85	1.73	4.06	2.33	1.78	3.02	3.44	5.78	6.18	1.66	4.4	2.62
11	16	1.55	2.84	3.48	4.88	5.76	1.93	4.21	2.28	1.52	2.88	3.56	5.58	5.88	2.04	4.36	2.84
12	17	1.41	2.64	3.74	5.46	5.58	2.33	4.17	1.84	1.54	2.68	3.58	5.43	5.68	2.04	4.14	2.6
13	17	1.48	3.02	3.94	4.68	5.21	2.46	3.73	1.27	1.56	2.58	3.69	4.48	5.35	2.13	3.79	2.23
14	17	1.61	2.34	3.48	4.28	5.58	1.87	3.97	2.1	1.72	2.58	3.88	5.16	5.36	2.16	3.64	1.92
15	17	1.56	3.02	3.48	4.92	5.21	1.92	3.65	1.73	1.92	2.73	3.48	4.68	5.24	1.56	3.32	1.4
16	18	1.6	2.56	3.66	5.34	5.42	2.06	3.82	1.76	1.58	2.84	3.58	5.54	5.66	2	4.08	2.5
17	18	1.44	2.76	3.76	4.86	5.56	2.32	4.12	1.8	1.58	2.66	3.62	4.38	5.74	2.04	4.16	2.58
18	18	1.42	2.7	3.86	5.24	5.46	2.44	4.04	1.6	1.94	2.58	3.56	4.56	5.28	1.62	3.34	1.4
19	18	1.56	2.48	3.68	4.59	5.64	2.12	4.08	1.96	1.48	2.68	3.52	5.68	5.88	2.04	4.4	2.92
20	18	1.64	2.58	3.56	4.64	5.42	1.92	3.78	1.86	1.64	2.66	3.72	4.48	5.68	2.08	4.04	2.4
21	15	1.68	2.64	3.54	5.56	5.72	1.86	4.04	2.18	1.62	2.68	3.48	4.52	5.56	1.86	3.94	2.32
22	16	1.68	2.68	3.66	5.28	5.86	1.98	4.18	2.2	1.59	2.78	2.52	4.38	5.72	0.93	4.13	2.54
23	16	1.56	2.54	3.72	4.78	5.68	2.16	4.12	1.96	1.68	2.46	3.56	5.48	5.48	1.88	3.8	2.12
24	19	1.68	2.46	3.62	5.56	5.64	1.94	3.96	2.02	1.64	2.68	3.78	4.68	5.78	2.14	4.14	2.5
25	19	1.52	2.44	3.44	4.56	5.58	1.92	4.06	2.14	1.62	2.66	3.62	5.66	5.84	2	4.22	2.6

PROFORMA

Name: Age: Sex:

Address: Phone:

Education:

Duration of visual challenge: Cause:

Visual acuity: Right: Left:

Using Braille: Yes / No Duration: Years

Hands: Both / Right / Left

ASSOCIATED SYMPTOMS:

Auditory / smell / taste / touch

Nerve conduction study:

Motor conduction study:

	Latency(m sec)		Amplitude(micro volt)		Area		Ncv m/sec	f min.
	Distal	Proximal	Distal	Proximal	Distal	proximal		
Right median								
Left median								
Right ulnar								
Left ulnar								
Right peroneal								
Let peroneal								
Right tibial								
Left tibial								

Sensory conduction study :

	Latency(m sec)	Amplitude(micro volts)	Ncv(m/sec)
Right median			
Left median			
Right ulnar			
Left ulnar			
Right sural			
Left sural			

Somatosensory evoked potential:

	N9(m sec)	N13 (m sec)	N20(m sec)
Right median			
Left median			

Brainstem Auditory Evoked Potential:

wave Latency (m sec)	I	II	III	IV	V
Right					
Left					

Inter Peak latency:

	Latency (m sec)
I – III	
III – V	
I - V	

INFORMATION SHEET

- ✓ We are conducting a study S **NERVE CONDUCTION STUDY & EVOKED POTENTIALS IN VISUALLY CHALLENGED PERSONS**

- ✓ The purpose of this study is to analyse the somato sensory response in visually challenged and compared with normal persons

- ✓ The privacy of the patients in the research will be maintained throughout the study. In the event of any publication or presentation resulting from the research, no personally identifiable information will be shared.

- ✓ Taking part in this study is voluntary. You are free to decide whether to participate in this study or to withdraw at any time; your decision will not result in any loss of benefits to which you are otherwise entitled.

- ✓ The results may be intimated to you at the end of the study period or during the study if anything is found abnormal which may aid in the management or treatment.

Signature of Investigator Signature of Participant
Date:

ஆராய்ச்சி தகவல் தாள்

இராஜீவ் காந்தி அரசு பொது மருத்துவமனைக்கு வரும் பார்வை குறைபாடுள்ள மாற்றுதிறனாளிகளிடம் இந்த ஆய்வு நடைபெறுகிறது.

பார்வை குறைபாடுள்ள மாற்றுதிறனாளிகளிடம் நரம்புகள் கடத்தும் திறன் குறித்து அறிவதே இந்த ஆய்வின் நோக்கமாகும்.

முடிவுகளை அல்லது கருத்துகளை வெளியிடும் போதோ அல்லது ஆராய்ச்சியின் போதோ தங்களது பெயரையோ அல்லது அடையாளங்களையோ வெளியிட மாட்டோம் என்பதையும் தெரிவித்துக் கொள்கிறோம்.

இந்த ஆராய்ச்சியில் பங்கேற்பது தங்களுடைய விருப்பத்தின் பேரில் தான் இருக்கிறது. மேலும் நீங்கள் எந்நேரமும் இந்த ஆராய்ச்சியிலிருந்து பின் வாங்கலாம் என்பதையும் தெரிவித்துக் கொள்கிறோம்.

இந்த சிறப்புப் பரிசோதனைகளின் முடிவுகளை ஆராய்ச்சியின் போது அல்லது ஆராய்ச்சியின் முடிவின்போது தங்களுக்கு அறிவிக்கப்படும் என்பதையும் தெரிவித்துக் கொள்கிறோம்.

ஆராய்ச்சியாளர் கையொப்பம் பங்கேற்பாளர் கையொப்பம்

தேதி :

PATIENT CONSENT FORM

Study Details : To study NERVE CONDUCTION & EVOKED POTENTIALS IN VISUALLY CHALLENGED PERSONS
Study Centre : Rajiv Gandhi Government General Hospital, Madras Medical College, Chennai - 600 003.

Patient may check (√) these boxes:

I confirm that I have understood the purpose of procedure for the above study. I have the opportunity to ask question and all my questions and doubts have been answered to my complete satisfaction. ☐

I understand that my participation in the study is voluntary and that I am free to withdraw at any time without giving reason, without my legal rights being affected. ☐

I understand that sponsor of the clinical study, others working on the sponsor's behalf, the ethical committee and the regulatory authorities will not need my permission to look at my health records, both in respect of current study and any further research that may be conducted in relation to it, even if I withdraw from the study I agree to this access. However, I understand that my identity will not be revealed in any information released to third parties or published, unless as required under the law. I agree not to restrict the use of any data or results that arise from this study. ☐

I agree to take part in the above study and to comply with the instructions given during the study and faithfully cooperate with the study team and to immediately inform the study staff if I suffer from any deterioration in my health or well being or any unexpected or unusual symptoms. ☐

I hereby give permission to undergo complete clinical examination and diagnostic tests including Electrophysiological examination ☐

I hereby consent to participate in this study. ☐
Read by:

Signature / Thumb impression:

Patient Name and Address: Place: Date:

Signature of Investigator:

Study Investigator's Name: Place : Date :

சுய ஒப்புதல் படிவம்

ஆய்வு செய்யப்படும் தலைப்பு

பார்வை குறைபாடு உள்ள மாற்று திறனாளிகளிடம் நரம்புகளின் கடத்தும் திறன் சம்பந்தமான ஆய்வு.

ஆராய்ச்சி நிலையம் : நரம்பியல் துறை,
இராஜீவ் காந்தி அரசு பொது மருத்துவமனை மற்றும்
சென்னை மருத்துவக் கல்லூரி,
சென்னை - 600 003.

பங்கு பெறுபவரின் பெயர் : உறவுமுறை :

பங்கு பெறுபவரின் எண். :

பங்கு பெறுபவர் இதனை (✓) குறிக்கவும்

மேலே குறிப்பிட்டுள்ள மருத்துவ ஆய்வின் விவரங்கள் எனக்கு விளக்கப்பட்டது. என்னுடைய சந்தேகங்களை கேட்கவும், அதற்கான தகுந்த விளக்கங்களைப் பெறவும் வாய்ப்பளிக்கப்பட்டது. ☐

நான் இவ்வாய்வில் தன்னிச்சையாகத்தான் பங்கேற்கிறேன். எந்தக் காரணத்தினாலோ எந்தக் கட்டத்திலும் எந்த சட்ட சிக்கலுக்கும் உட்படாமல் நான் இவ்வாய்வில் இருந்து விலகிக் கொள்ளலாம் என்றும் அறிந்து கொண்டேன். ☐

இந்த ஆய்வு சம்மந்தமாகவோ, இதை சார்ந்த மேலும் ஆய்வு மேற்கொள்ளும்போதும் இந்த ஆய்வில் பங்குபெறும் மருத்துவர் என்னுடைய மருத்துவ அறிக்கைகளைப் பார்ப்பதற்கு என் அனுமதி தேவையில்லை என அறிந்து கொள்கிறேன். நான் ஆய்வில் இருந்து விலகிக் கொண்டாலும் இது பொருந்தும் என அறிகிறேன். ☐

இந்த ஆய்வின் மூலம் கிடைக்கும் தகவல்களையும், பரிசோதனை முடிவுகளையும் மற்றும் சிகிச்சை தொடர்பான தகவல்களையும் மருத்துவர் மேற்கொள்ளும் ஆய்வில் பயன்படுத்திக் கொள்ளவும் அதைப் பிரசுரிக்கவும் என் முழு மனதுடன் சம்மதிக்கிறேன். ☐

இந்த ஆய்வில் பங்கு கொள்ள ஒப்புக்கொள்கிறேன். எனக்குக் கொடுக்கப்பட்ட அறிவுரைகளின்படி நடந்து கொள்வதுடன், இந்த ஆய்வை மேற்கொள்ளும் மருத்துவ அணிக்கு உண்மையுடன் இருப்பேன் என்றும் உறுதியளிக்கிறேன். என் உடல் நலம் பாதிக்கப்பட்டாலோ அல்லாத எதிர்பாராத வழக்கத்திற்கு நோய்க்குறி தென்பட்டாலோ உடனே அதை மருத்துவ அணியிடம் தெரிவிப்பேன் என உறுதி அளிக்கிறேன். ☐

இந்த ஆய்வில் எனக்கு நரம்புகள் கடத்தும் திறன் குறித்த பரிசோதனைகள் செய்து கொள்ள நான் முழு மனதுடன் சம்மதிக்கிறேன். ☐

பங்கேற்பவரின் கையொப்பம்..... இடம்..... தேதி
கட்டைவிரல் ரேகை

பங்கேற்பவரின் பெயர் மற்றும் விலாசம்.....

ஆய்வாளரின் கையொப்பம்..... இடம்..... தேதி

ஆய்வாளரின் பெயர்.....

INSTITUTIONAL ETHICS COMMITTEE
MADRAS MEDICAL COLLEGE, CHENNAI -3

Telephone No : 044 25305301
Fax : 044 25363970

CERTIFICATE OF APPROVAL

To
Dr. V. Sadeesh Kumar
PG in DM Neurology
Madras Medical College, Chennai -3

Dear Dr. V. Sadeesh Kumar

The Institutional Ethics committee of Madras Medical College, reviewed and discussed your application for approval of the proposal entitled "Genetic analysis in neuro muscular disease patients" No.36042012.

The following members of Ethics Committee were present in the meeting held on 19.04.2012 conducted at Madras Medical College, Chennai -3.

1.	Dr. S.K. Rajan. M.D.,FRCP.,DSc	-- Chairperson
2.	Prof. Pregna B. Dolia MD Director , Institute of Biochemistry, MMC, Ch-3	-- Member Secretary
3.	Prof. B. Kalaiselvi MD Prof. of Pharmacology ,MMC, Ch-3	-- Member
4.	Prof. C. Rajendiran, MD Director , Inst. of Internal Medicine, MMC, Ch-3	-- Member
5.	Prof. Md. Ali. MD.DM Prof & HOD, Dept. of MGE, MMC, Ch-3	-- Member
6.	Prof.P.Karkuzhali MD Director i/c, Prof., Inst. of Pathology, MMC, Ch-3	-- Member
7.	Prof. S. Deivanayagam MS Prof of Surgery, MMC, Ch-3	-- Member
8.	Prof. A. Radhakrishnan MD Prof of Internal Medicine, MMC, Ch-3	-- Member
9.	Thiru. S. Govindsamy. BABL	-- Lawyer
10.	Tmt. Arnold Soulina MA MSW	-- Social Scientist

We approve the proposal to be conducted in its presented form.

Sd/ Chairman & Other Members

The Institutional Ethics Committee expects to be informed about the progress of the study, and SAE occurring in the course of the study, any changes in the protocol and patients information / informed consent and asks to be provided a copy of the final report.

Member Secretary, Ethics Committee

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E-mail	sadeesh1993@gmail.com
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First 100 words of your submission

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